CHAPTERVI

PLASTICSURGERY

1. Bed sore
2. Burn
3. Skin tumor
4. Soft tissues infection

BEDSORE/ PRESSURE ULCERS

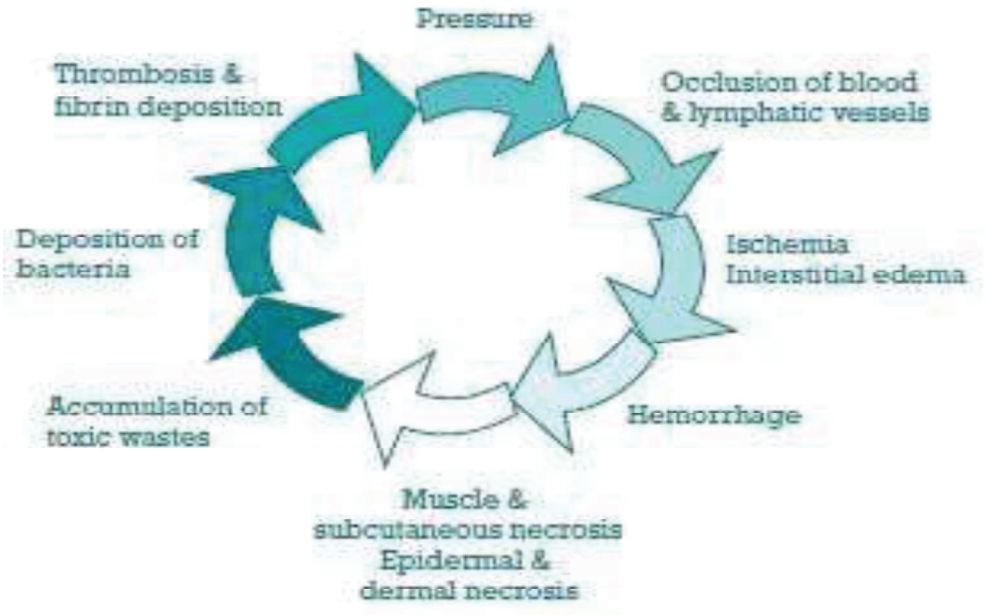
KY ChanmonyRaksmey, Tep Borin

I. DEFINITION

- Any lesion caused by unrelieved pressure that results in damage to underlying tissue
- Usually occurs over a bony prominence
- Staged to classify the degree of tissue damage observed (National Pressure Ulcer Advisory Panel, 1989)

II. CAUSES

- Area of greatest damage near bony prominences



(Goode & Allman, 1989)

- By the time inflammation becomes visible, necrosis of muscle, fat and subcutaneous tissue may have occurred
- Worsen between 1-2 days
- Underlying damage has begun as tissue destruction has been set in motion (Patterson & Bennett, 1995)

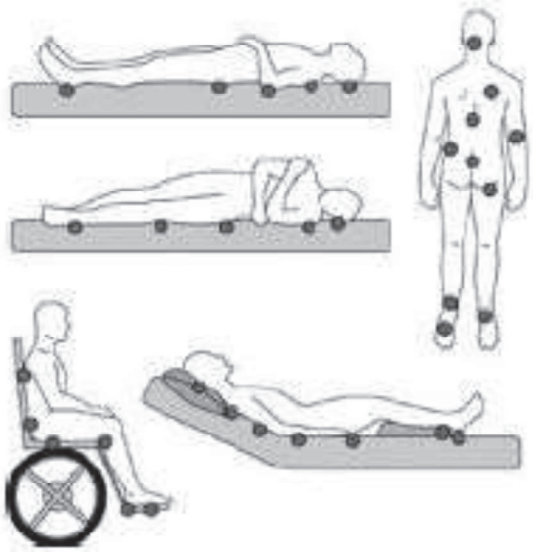
a) Risk Factors for Developing Pressure Ulcers

1. Mobility
2. Activity
3. Sensory Perception
4. Nutrition
5. Arteriolar Pressure
6. Pressure
7. Moisture
8. Friction
9. Shear
10. Age

(Braden, 1987)

b) Pressure Ulcer Sites

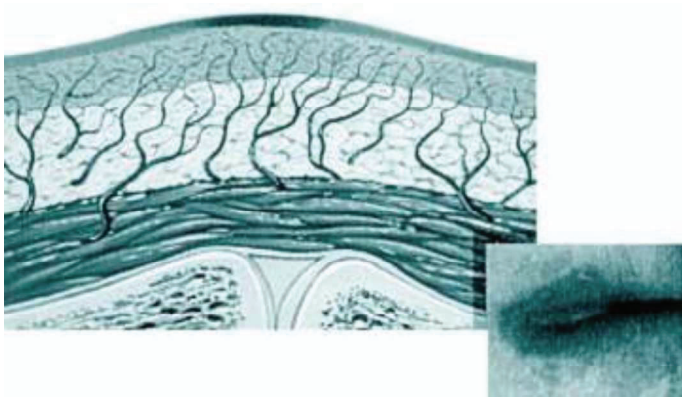
1. Supine Position: heels, sacrum, elbows, scapulae, back of head
2. Lateral Position: malleous, medial and lateral condyles, greater trochanter, ribs, acromion process, ear
3. Prone Position: toes, knees, genitalia (men), breasts (women), acromion process, cheek and ear
4. Sitting Position: elbow, sacrum, ischium



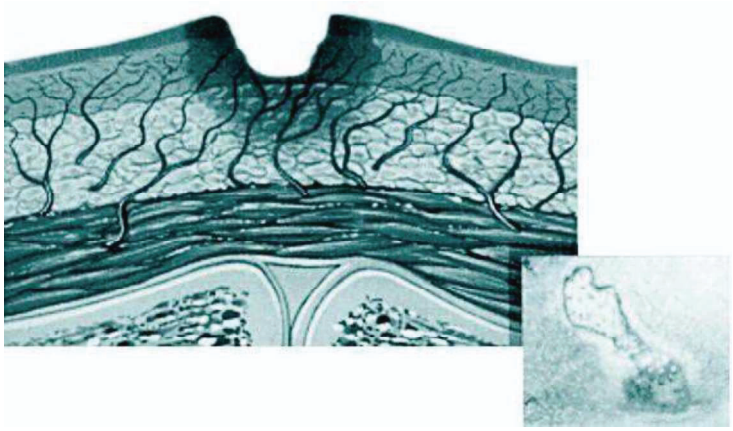
c) Pressure Ulcer Staging System

The HSE Wound Management Guideline (2018) 4 describes the following Pressure Ulcer Staging System (see Appendix 1): 4 HSE

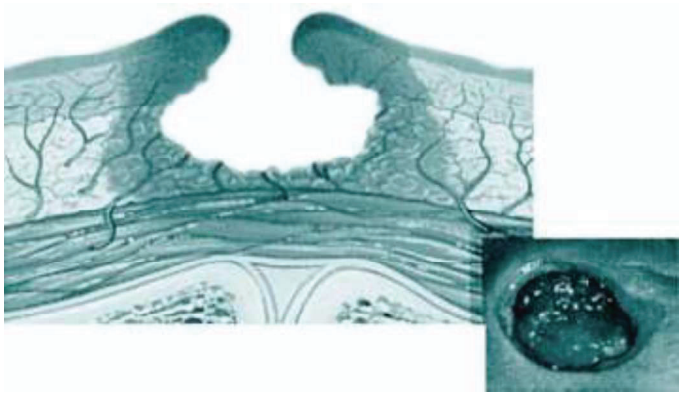
- Stage I: Intact skin with non – blanchable redness of a localised area usually over a bony prominence. Discolouration of the skin, warmth, oedema, hardness or pain may also be present. Darkly pigmented skin may not have visible blanching. The area may be painful, firm, soft, warmer or cooler as compared to adjacent skin.



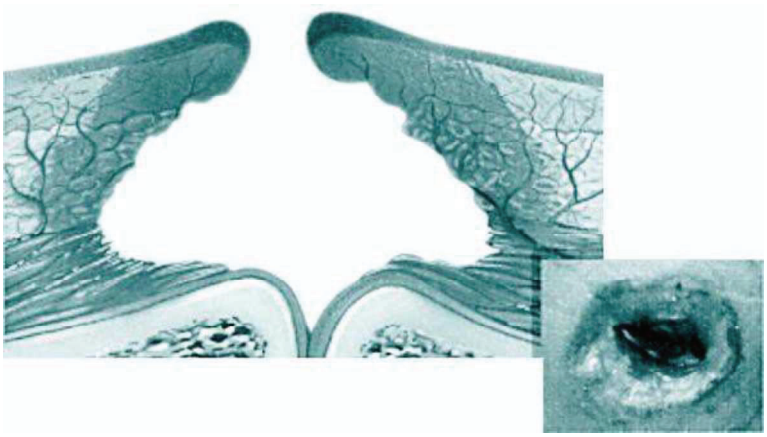
- Stage II: Partial thickness skin loss of dermis presenting as a shallow ulcer with a red pink wound bed, without slough. May present as an intact or open/ ruptured serum filled blister filled with serous or sero-sanguineous fluid. Presents as a shiny or dry shallow ulcer without slough or bruising.



- Stage III: Full thickness skin loss. Subcutaneous fat may be visible but bone, tendon or muscles are not exposed. Slough may be present but does not obscure the depth of tissue loss. The stage may include undermining or tunnelling.



- Stage IV: Full thickness tissue loss with exposed bone, tendon or muscle. Slough or eschar may be present. This stage often includes undermining and tunnelling. Exposed bone / muscle is visible or directly palpable.



Suspected deep pressure and shear induced tissue damage, depth unknown. For Service Users with non-blanchable redness and purple/maroon discoloration of intact skin combined with a history of prolonged, unrelieved pressure/shear, this skin change may be an indication of emerging, more severe pressure ulceration i.e. an emerging Stage III or IV Pressure Ulcer. Clear recording of the exact nature of the visible skin changes, including recording of the risk that these changes may be an indication of emerging more severe pressure ulceration, should be documented in the Service User's health record. These observations should be recorded in tandem with information pertaining to the Service User's history of prolonged, unrelieved pressure/shear.

It is estimated that it could take 3-10 days from the initial insult causing the damage, to become a Stage III or IV Pressure Ulcer. Stable eschar (dry adherent, intact without erythema or fluctuance) on the heel serves as the body's biological cover and should not be removed. It should be documented as at least Category / Stage III until proven otherwise.

See Appendix 1 for illustration of the HSE Pressure Ulcer Category/Staging System Recommendations.

The Braden Scale for Predicting Pressure Sore Risk

Educational Workshop for RNs and RPNs: Assessment and Management of Pressure Ulcers

Assessing Risk Factors for Developing Pressure Ulcers

The Braden Scale for Predicting Pressure Sore Risk

Sensory Perception Ability to respond meaningfully to pressure-related discomfort.	1. Completely limited Unresponsive (does not moan, flinch or grasp) to painful stimuli due to diminished level of consciousness or sedation, or limited ability to feel pain over most of body surface.	2. Very limited Responds only to painful stimuli. Cannot communicate discomfort except by moaning or restlessness, or has a sensory impairment that limits the ability to feel pain or discomfort over half of body.	3. Slightly limited Responds to verbal commands but cannot always communicate discomfort or need to be turned, or has some sensory impairment that limits ability to feel pain or discomfort in 1 or 2 extremities.	4. No impairment Responds to verbal commands; has no sensory deficit that would limit ability to feel or voice pain or discomfort.	
Moisture Degree to which skin is exposed to moisture.	1. Constantly moist Skin is kept moist almost constantly by perspiration, urine etc. Dampness is detected every time patient is moved or turned.	2. Very moist Skin is often, but not always, moist. Linen must be changed at least once a shift.	3. Occasionally moist Skin is occasionally moist, requiring an extra linen change approximately once a day.	4. Rarely moist Skin is usually dry, linen only requires changing at routine intervals.	
Activity Degree of physical activity.	1. Bedfast Confined to bed.	2. Chairfast Ability to walk severely limited or non-existent. Cannot bear own weight and/or must be assisted into chair or wheelchair.	3. Walks occasionally Walks occasionally during day, but for very short distances with or without assistance. Spends majority of each shift in bed or chair.	4. Walks frequently Walks outside the room at least twice a day and inside room at least every 2 hours during waking hours.	
Mobility Ability to change and control body position.	1. Completely immobile Does not make even slight changes in body or extremity position without assistance.	2. Very limited Makes occasional, slight changes in body or extremity position but unable to make frequent or significant changes independently.	3. Slightly limited Makes frequent though slight changes in body or extremity position independently.	4. Walks frequently Makes major and frequent changes in position without assistance.	
Nutrition Usual food intake pattern.	1. Very poor Never eats a complete meal. Rarely eats more than 1/3 of any food offered. Eats 2 servings or less of protein (meat or dairy products) per day. Takes fluids poorly. Does not take a liquid dietary supplement, or is NPO and/or maintained on clear liquids or IVs for more than 5 days.	2. Probably inadequate Rarely eats a complete meal and generally eats only about half of any food offered. Protein intake includes only 3 servings of meat or dairy products per day. Occasionally will take a dietary supplement, or receives less than optimum amount of liquid diet or tube feeding.	3. Adequate Eats over half of most meals. Eats a total of 4 servings of protein (meat, dairy products) each day. Occasionally will refuse a meal, but will usually take a supplement if offered, or is on a tube feeding or TPN regimen, which meets most of nutritional needs.	4. Excellent Eats most of every meal. Never refuses a meal. Usually eats a total of 4 or more servings of meat and dairy products. Occasionally eats between meals. Does not require supplementation.	
Friction and Shear	1. Problem Requires moderate to maximum assistance in moving. Complete lifting without sliding against sheets is impossible. Frequently slides down in bed or chair, requiring frequent repositioning with maximum assistance. Spasticity, contractures or agitation lead to almost constant friction.	2. Potential Problem Moves feebly or requires minimum assistance. During a move skin probably slides to some extent against sheets, chair restraints, or other devices. Maintains relatively good position in chair or bed most of the time but occasionally slides down.	3. No apparent problem Moves in bed and in chair independently and has sufficient muscle strength to lift up completely during move. Maintains good position in bed or chair at all times.	NOTE: Patients with a total score of 16 or less are considered to be at risk of developing pressure ulcers. (15 or 16=low risk; 13 or 14=moderate risk; 12 or less=high risk)	

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Nursing Best Practice Guidelines Program

Registered Nurses' Association of Ontario

III. DIAGNOSTIC PROCEDURE

History and Physical Examinations:

Pressure ulcers should be assessed in the context of the patient/resident's overall physical and psychological health.

i. Physical health:

Intrinsic Factors – Relate to the aspects of the client's physical, medical or psychosocial condition.

- Physical
- Nutritional status
- Reduced mobility/immobility
- Posture/contractures
- Repetitive stress syndrome
- Neurological/sensory impairment
- Incontinence
- Age
- Level of consciousness
- Medical
- Acute illness
- History of previous pressure damage
- Vascular disease
- Chronic/terminal illness
- Psychosocial
- Stress and anxiety
- Sleep disturbances

Extrinsic Factors – Are derived from the environment

- Friction
- Pressure
- Shearing
- Hygiene
- Living Conditions
- Medication
- Garments
- Transfer slings
- Restraint use

- Support systems for pressure relief

ii. Psychological health:

- Mental Status
- Learning Ability
- Depression
- Social Support
- Polypharmacy/Overmedication
- Alcohol/Drug Use
- Goals/Values and Lifestyle
- Sexuality
- Culture and Ethnicity
- Pain as a symptom
- Stressors
- Resources (availability and skill of caregivers, finances, equipment) of individuals being treated for pressure ulcers in the home

iii. Quality of Life

- How do pressure ulcers affect the patient's quality of life?
- Ask patient to describe his/her current health status
- Ask patient how the pressure ulcer impacts on his/her day to day living

iv. Pressure Ulcer Assessment

- Location
- Depth/Stage
- Size (cm)
- Odour
- Sinus Tracts
- Undermining
- Tunnelling
- Exudate
- Wound Bed
- Appearance/Condition of Surrounding Skin (Periwound) and Wound Edges
-

v. Location (Barton & Parslow, 1996)

Most common sites: Sacrum (60% of all sores). Ischial Tuberosities in the sitting patient, greater trochanter, heel (15%)

Uncommon Sites: Elbow. Knee. Ankle. Occiput

vi. Measurement

- Size (how to measure)
- Use a ruler, transparency tracings or photography
- Measure the width/depth/breadth
- Weekly measurement are usually suffi cient
- Depth
- Sinus Tracts
- Undermining (clock measurement)
- Tunnelling

vii. Exudate

- Type
  - *Serous:* Clear fl uid with visual absence of blood, pus or other debris
  - *Sanguinous: Bloody, appearing to be composed entirely of blood*
  - *Serosanguinous: Blood mixed with obvious quantities of clear fluid*
  - *Purulent: Pus* like in appearance, cloudy and viscous
- Amount
  - *Dry: Wound does not produce exudates*
  - *Low: Wound is moist*
  - *Moderate: Surrounding skin is wet*
  - *High:*
    - Surrounding skin is saturated (sometimes macerated)
    - Wound is bathed in fluid

viii. Wound Base

black eschar, necrotic cleanse/debride yellow fibrin or slough cleanse/debride pink/red granulation protect

IV. THERAPEUTIC APPROACH

v. Stabilize patient

Prevent pressure and trauma in order to maintain skin integrity

- **Do's**

1. Prevent local areas of pressure

- **Don'ts**

1. Prevent local areas of pressure
2. Provide pressure reduction via ues of mattress overlays, cushions, or foams

- Prevent maceration imitation and bacterial growth

1. Keep skin hydrated and supple
2. Keep skin clean and dry
3. Wash skin gently with water; pH balanced soaps or skin cleansers
4. Investigate and manage incontinence

- Prevent moisture retention and excessive warmth

1. Avoid use of plastics (under pads and diapers) choose liner or fabric instead
2. Increase vigilance when patient is diaphoretic
3. Protect skin by applying barrier creams, gels or pastes
4. Avoid applying lotion between toes

- Involvement of Physicians and Surgeons

1. General Practitioners
2. Geriatricians
3. Dermatologists
4. Plastic Surgeons
5. Vascular Surgeons

vi. Empirical treatment

Wound Healing: A cascade of events of the biologic and immunologic system

(CREST, 1998). The recognized end point in healing is total wound closure (Robson et al., 1998).

A. Mechanical Impediment:

- Dead tissue in a wound slows wound healing by impeding the migration of epithelial cells from wound edges
- Eschar prevents the wound edges from drawing together and is a breeding ground for bacteria
- Foreign material (lint and pieces of dressing) can impede epithelial migration and increase likelihood of infection

- A dry wound site can impede the viability of the cells and tissues involved in wound healing When applied to healthy tissue, some antimicrobial preparations and cleansing agents may delay wound healing and may even be toxic to viable tissue

Example: Full-strength hydrogen peroxide can damage newly forming cells that remain in the wound. To reduce surface bacteria and tissue trauma, irrigate the wound gently with 100 to 150 ml of normal saline.

Use enough irrigation pressure to enhance wound cleansing without causing trauma to the wound bed. 4-15 psi is the safe and effective irrigation pressure range. To achieve 4-15 psi, use a 20-35 ml syringe with a 19 gauge angiocath.

B. Reminder:

Do not use skin cleansers or antiseptics to clean the wound. For example:

1. Povidine Iodine (Betadine)
2. Iodophor
3. Sodium Hypochlorite Solution
4. Hydrogen Peroxide
5. Acetic Acid

C. Debridement:

Debridement is often necessary to remove devitalized tissue and exudates, reduce the risk of infection, prepare the wound bed and promote healing. Debridement can be:

- Autolytic
- Mechanical
- Sharp
- Enzymatic

D. Assessment and Management of Infected Wounds:

a) Infection is diagnosed when >105 bacterial/gram tissue is present

b) Clinical Signs of Infection

- Delayed healing/dehiscence
- Increased wound pain
- Malodour
- Abscess/sinus formation
- Localized swelling/redness/heat
- Increased level of exudates/purulent discharge

- Pyrexia, rigours
- Tachycardia

E. Antibiotics:

- Systemic antibiotics are not required for pressure ulcers with only clinical signs of local infection
- Exceptions with locally infected wounds requiring systemic antibiotics
- Systemic antibiotics are used when the virulence of the organism is high and the host's defenses are compromised

F. Antiseptics:

- Use of cytotoxic antiseptics to reduce bacteria in wound tissue is not recommended
- Typical management of infected wounds includes the use of topical antimicrobials rather than antibiotics or antiseptics

G. Dressings

- Sterile dressings should be used in all care settings
- Avoid all occlusive dressings if anaerobic infection is suspected or cultured
- Protect non-infected ulcers with occlusive dressings

vii. Directed treatment

Stage I **Goals:** To reduce further skin breakdown and prevent skin loss. Protect against moisture and friction

- **Interventions:** Protect area from friction, shear, and maceration using a transparent film dressing or
- thin hydrocolloids. Provide pressure relieving devices to reduce friction and shearing forces
- **Goals:** To reduce further skin breakdown and prevent skin loss. To protect the surrounding skin from moisture by managing exudates and providing a moist wound environment to promote healing.
- **Interventions:** Clean the ulcer with normal saline. Protect the wound by covering it with a transparent dressing or hydrocolloid. For moderate amount of exudates, use an absorbent foam dressing. Use liquid or solid barriers to protect periwound skin from maceration damage.

Stage II

Stage III & IV

- **Goals:** To remove cell debris and promote autolysis. To provide clean, moist environment for the healing process to begin. To absorb exudates. To protect from contamination and trauma. To decrease dressing changes. To protect surrounding skin
- **Interventions:**
- **Dry Cavity:** Irrigate with normal saline using a 20-35 ml syringe and 19-gauge needle or angiocath. Protect periwound skin with a protective barrier.

  Fill dead space with appropriate filler (including sinus tracts). Line cavity with gel and place 4 x 4 gauze packed loosely. Protect from contamination by use of an absorbent outer semi-occlusive dressing
- **Interventions: Exudating Cavity:** Irrigate with normal saline using a 20- 35 ml syringe and 19-gauge needle or angiocath. Protect periwound skin using a protective barrier. Fill dead space with appropriate filler (including sinus tracts). Use absorbent foam dressings. Protect from contamination by use of an outer semi-occlusive dressing.

Surgery:

- **Goals:** To debride and remove dead tissue. To rehydrate the eschar by providing a clean, moist
- environment for the healing process to begin. To promote closure/healing
- **Interventions:** Clean with normal saline. Surgical debridement by MD or trained person. Autolytic debridement using gels. Protect

  periwound skin using a protective barrier. Cover with transparent dressing.
- **Antiseptics:** cytotoxic antiseptics is not recommended. Typical management: topical antimicrobials rather than antibiotics or antiseptics
- **Dressings:** Sterile dressings should be used in all care settings. Avoid all occlusive dressings if anaerobic infection is suspected or cultured. Protect non-infected ulcers with occlusive dressings.
- **Pain Assessment and Management Operative Repair of Pressure Ulcers:** Wounds can be closed by direct closure, skin grafting, skin flaps, musculocutaneous flaps and free flaps. Procedure is performed by MD and plastic surgeon. Candidates for operative repair are medically stable, adequately nourished, and can tolerate operative blood loss and postoperative immobility. Other considerations are quality of life, patient

preferences, treatment goals, risk of recurrence, and expected rehabilitative outcome

viii. Monitoring

- **PostoperativeCare: Havepatient slowly increaseperiods oftime sitting or lyingon theflap to increaseits toleranceto pressure. Monitoring the flap for pallor,redness,orboththatdonotresolvein10minutesor pressure relief.**
- **Discharge/Transfer of Care Arrangements: Patients/residents moving between care setting should** have the
  - following information provided:
  - Risk factors identified
  - Details of pressure points and skin condition prior to transfer
  - Type of bed/mattress required
  - Details of healed ulcers
  - Stage, site and size of existing ulcers
  - History of ulcers, previous treatments and dressings used
  - Type of dressing currently used and frequency of change
  - Allergies to dressing products
  - Need for on-going nutritional support

BURN

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I. DEFINITION

Burn is a tissue injury that results from thermal application (hot/cold) and from application of physical or chemical energy.

II. CAUSES

Burns are caused by dry heat, moist heat, cold injury, chemical burn, electrical burns, ionizing radiation and friction.

III. DIAGNOSTIC PROCEDURE

III.1. Clinical argument

III.1.A Histories

- Time of burn
- Histories medico-surgeries
- Last meal
- Events and environment of burn injury

III.2. III.1.B Degree of burn

1st degree: minor epithelial damage, present as redness, clinically insignificant 2nd degree: partial thickness burns and scalds. These burns are painful.

3rd degree: full thickness burn, usually painless.

4th degree: As the third but extends into fascia and/or muscle

Fig (1)

Depth of Burn	Appearance	Sensation
First degree	Red Blanches with pressure Dry **No** blisters	Painful
Second degree *Partial thickness* - **superficial**	Red Blanches with pressure Moist, weeping Blisters	Painful to temperature and air
Second degree *Partial thickness* - **deep**	Variable colour **No** blanching with pressure Wet, waxy or dry Blisters (easily unroofed)	Perceptive of pressure only
Third degree *Full thickness*	Waxy white, leathery gray, charred or black **No** blanching with pressure Dry, inelastic	Deep pressure only
Fourth degree	As with Third degree, but extends into fascia and/or muscle	Deep pressure

Fig 2

Depth of Burn	Appearance	Sensation
First degree	Red Blanches with pressure Dry **No** blisters	Painful
Second degree *Partial thickness* - **superficial**	Red Blanches with pressure Moist, weeping Blisters	Painful to temperature and air
Second degree *Partial thickness* - **deep**	Variable colour **No** blanching with pressure Wet, waxy or dry Blisters (easily unroofed)	Perceptive of pressure only
Third degree *Full thickness*	Waxy white, leathery gray, charred or black **No** blanching with pressure Dry, inelastic	Deep pressure only
Fourth degree	As with Third degree, but extends into fascia and/or muscle	Deep pressure

It is common to find all types of burns within the same wound and the depth may change with time, especially if infection occurs.

I.1.A. **Calculation of surface area** of burn Adults :

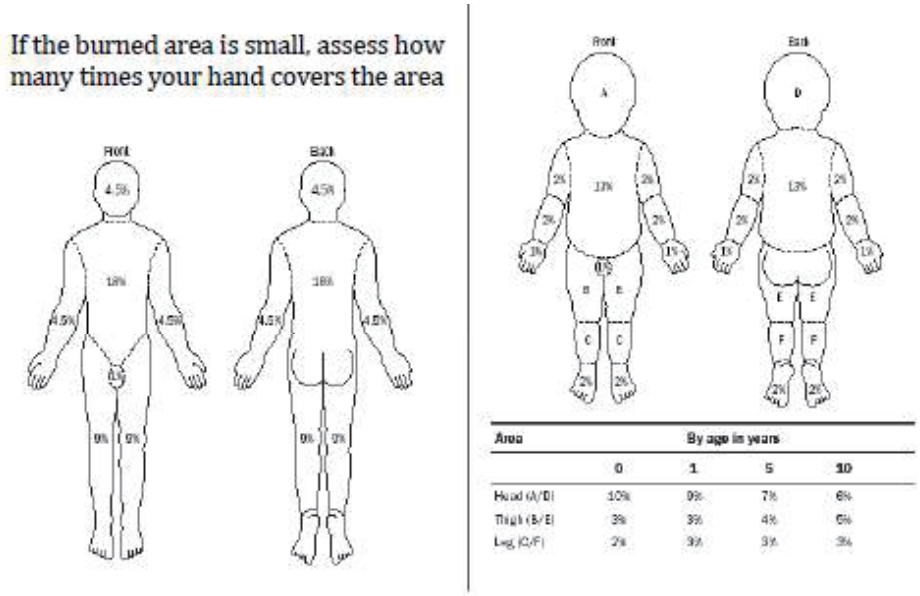
+ the Rull of 9 is commonly used to estimate the burned surface area in adults.
+ the body is divided into anatomical regions that represent 9% (or multiples of 9%) of the total body surface area.
+ the outstrectched palm and fingers approximate to 1%

Children: the rule of 9s is modified for infants and children since their heads and lower extremities represent different proportions of body surface area.

Technical procedure

Laboratory test: blood test Chest X-ray if necessary

Fig 3



III.2.Admission criteria

- Partial thickness burn of >25% of TBSA adults
- >20% burns in children below 10 years old and adults > 50 years old
- Full thickness burn > 10% TBSA
- Burn of face, eyes, ears, and perineum
- Chemical burns, electrical burns, inhalation injury, and underlying debilitating illness
- Moderate burns > 15% TBSA (TBSA=total body surface area)

IV. THERAPEUTIC APPROACH

- In all cases, administer tetanus (inj. TT 0.5cc IM) prophylaxis
- Remove with debride devitalized and loose tissue
- Pain management
- Airway: airway burns, smoke inhalation syndrome, severe burns of face and neck, laryngeal edema may need inhalation or tracheostomy
- O2 inhalation: started at 8-12 lit/min in patients having dyspnea, major burns, airway burns, and smoke inhalation syndrome
- Fluid resuscitation:
    - Insertion of large bore cannulae
    - oParkland formula is to be used for calculation or fluid requirement in first 24 hrsi.e 4ml/kg/% of burn.
    - The fluid of choice is Ringer lactate
    - 50% of the fluid is to be transfused in first 8hrs from the time of burns and the remaining 50% in next 16hrs
    - Adequate resuscitation is monitored by vital parameters and urine output of 0.5- 1ml/kg/hr.
    - oIf the urine output is in excess of 2ml/kg.hr the rate of infusion should be reduced
    - Next 24hrs:
        - Total volume = ½ of first day
        - Colloids (0.5ml/kg/%) and 5% glucose or isotonic glucose saline to make up the rest
- Nasogastric tube insertion if necessary
- Antibiotics:
    - Used of broad-spectrum antibiotics lead to colonization of wound by resistant organisms
    - However, Ceftazidime + Amikacin + Metronidazole canbe used after 48hrs pending sensitivity of organism isolated from the wound
- H2-receptor antagonists:
    - Acute upper GI erosions and ulcers (curling ulcer) may occur in patients with severe burn injuries. The common clinical finding in such patients is painless GI hemorrhage. Traitment of acute stress ulceration is principally preventive

- H2-receptor antagonists inhibit gastric acid secretion, this prophylaxis against acute stress ulceration is initiated immediately after admission. Commonly used drug is inj. Ranitidine 50mg x 8th hourly

- Nutrition (third day onwards)

oStart with oral fluids and Fortified milk:

- Calculation calories; Adults 25kcal x Kg + 40 x %burns; Child 60kcal x kg + 35 x %burns
- Protein; Adult 1gm/kg +3gm/%burns; Child 3gm/kg + 1gm/%burns
- Vitamin supplements

- Care of burn wounds:
  - Clean wound with dilute normal saline or ringer's lactate
  - Debride blisters and nonviable tissues
  - Use Silver sulphadiazine or Soframycine ointment to cover the wounds
  - Wounds can be managed by exposure method or by closed method by covering wounds with Vaseline gauze and bandages

- Others supportive therapy:
  - Physiotherapy for joints
  - Compression dressings to prevent hypertrophic scars
  - Psychotherapy
  - Rehabilitation therapy

V. COMPLICATIONS:

V.1. Early complications

- burn shock
- renal failure
- smoke inhalation syndrome
- septicemia

V.2. Late complications

- hypertrophic scars
- keloids
- contractures
- protein losing enteropathy

Appendix 2: recognising burn depths chart

Epidermal burn (erythema)

- Damage to epidermis only. Skin intact, no blisters present
- Erythema. Red
- Brisk capillary refill
- Heal spontaneously within 3–7 days with moisturiser or protective dressing

Superficial dermal burn

- Damage to upper layer of dermis
- Pink. Blisters present or absent
- Brisk capillary refill (under blister)
- Should heal within 7–10 days with minimal dressing requirements

Mid dermal burn

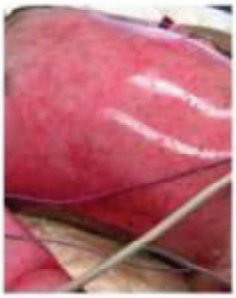
- Damage into mid dermis
- Dark pink
- Sluggish capillary refill
- Should heal within 14 days.
- Deeper areas may need surgical intervention and referral

Deep dermal burn

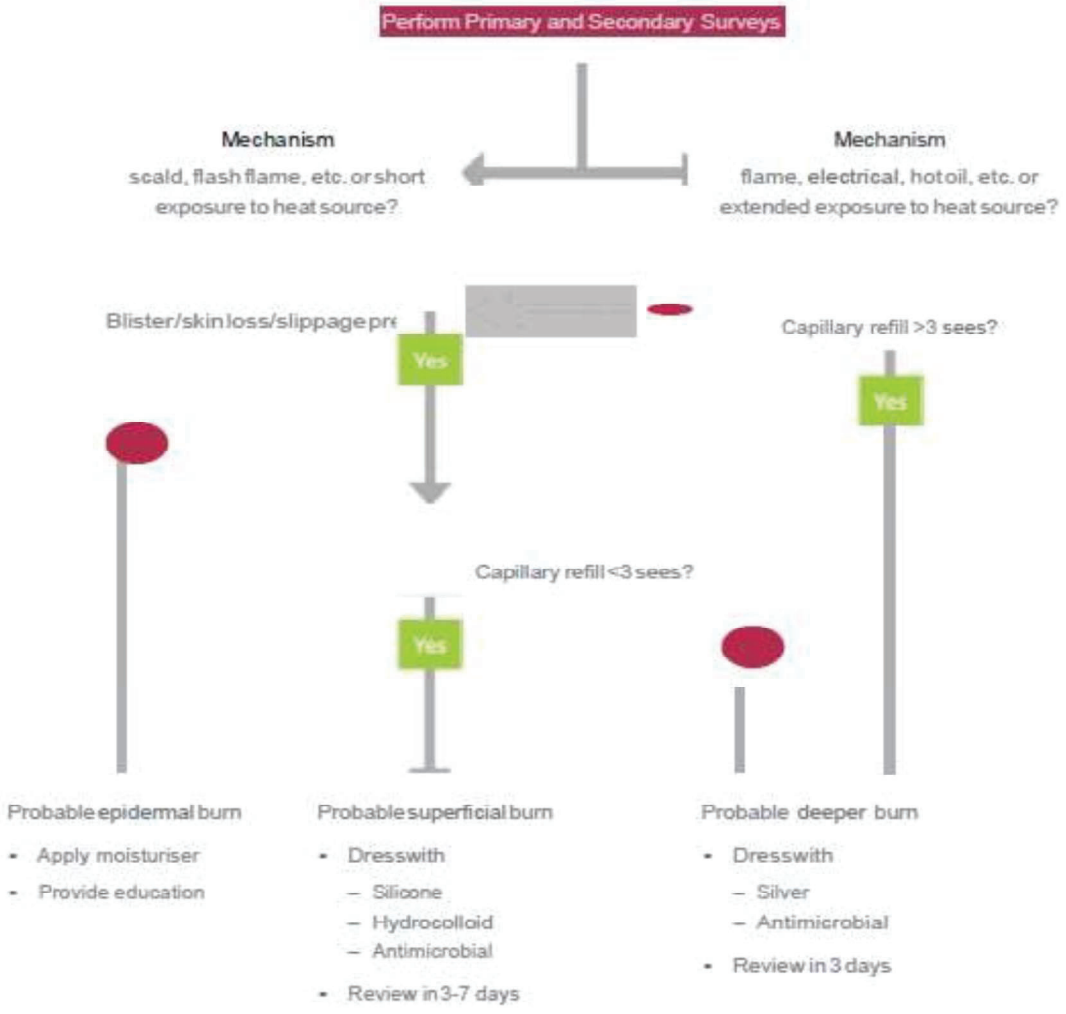
- Burn extends into the deeper layers of the dermis, but not through the entire dermis
- Blotchy red/white
- Sluggish to absent capillary refill
- Generally need surgical intervention.
- Refer to specialist unit

Full thickness burn

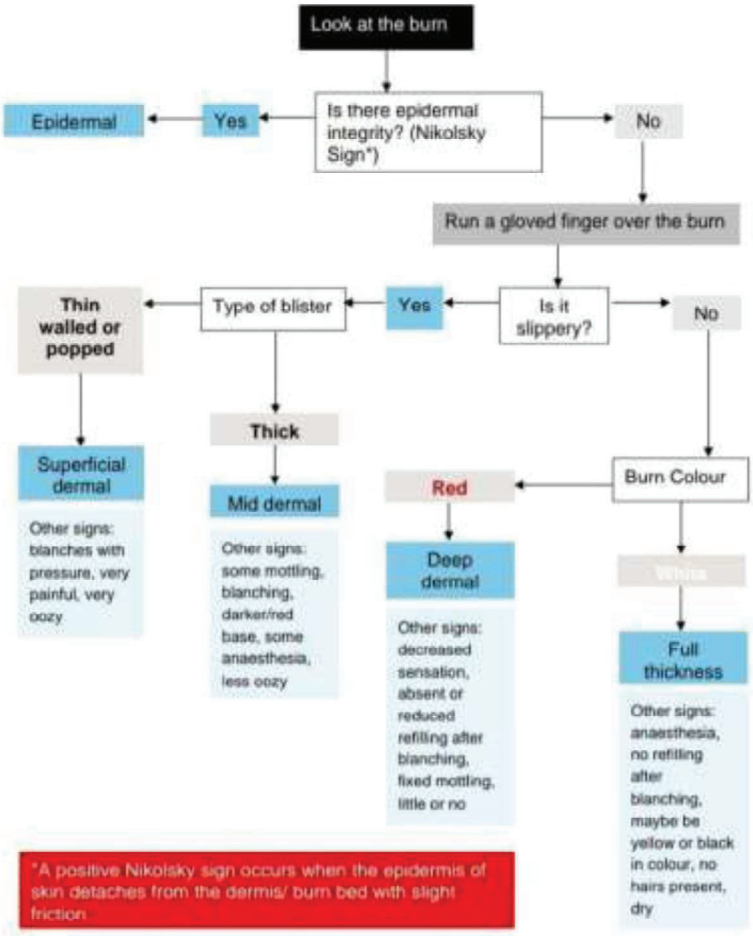
- Entire destruction of dermis, sometimes underlying tissue involved
- White, waxy, brown, black.
- No capillary refill
- Surgical intervention and long-term scar management required
- Refer to specialist unit



Burn patient dressing decision-making tree



Appendix I – Protocol for burn depth assessment


Look at the burn
Is there epidermal integrity? (Nikolsky Sign*)
Yes
Epidermal
No
Run a gloved finger over the burn
Is it slippery?
Yes
Type of blister
Thin walled or popped
Superficial dermal
Other signs: blanches with pressure, very painful, very oozy
Thick
Mid dermal
Other signs: some mottling, blanching, darker/red base, some anaesthesia, less oozy
No
Burn Colour
Red
Deep dermal
Other signs: decreased sensation, absent or reduced refilling after blanching, fixed mottling, little or no
Full thickness
Other signs: anaesthesia, no refilling after blanching, maybe be yellow or black in colour, no hairs present, dry
*A positive Nikolsky sign occurs when the epidermis of skin detaches from the dermis/ burn bed with slight friction

Appendix N – Management of facial burns

Initial care (may be done in theatre/technical suite)

- All non-viable tissue should be gently removed by picking and washing with gauze. Beard, moustache and sideburn hair will have been shaved completely and scalp hair similarly shaved away from the burn edge. Soft paraffin is applied. **Do not apply Flamazine- risk of corneal ulceration.**

Ongoing care (on ward, aseptic technique)

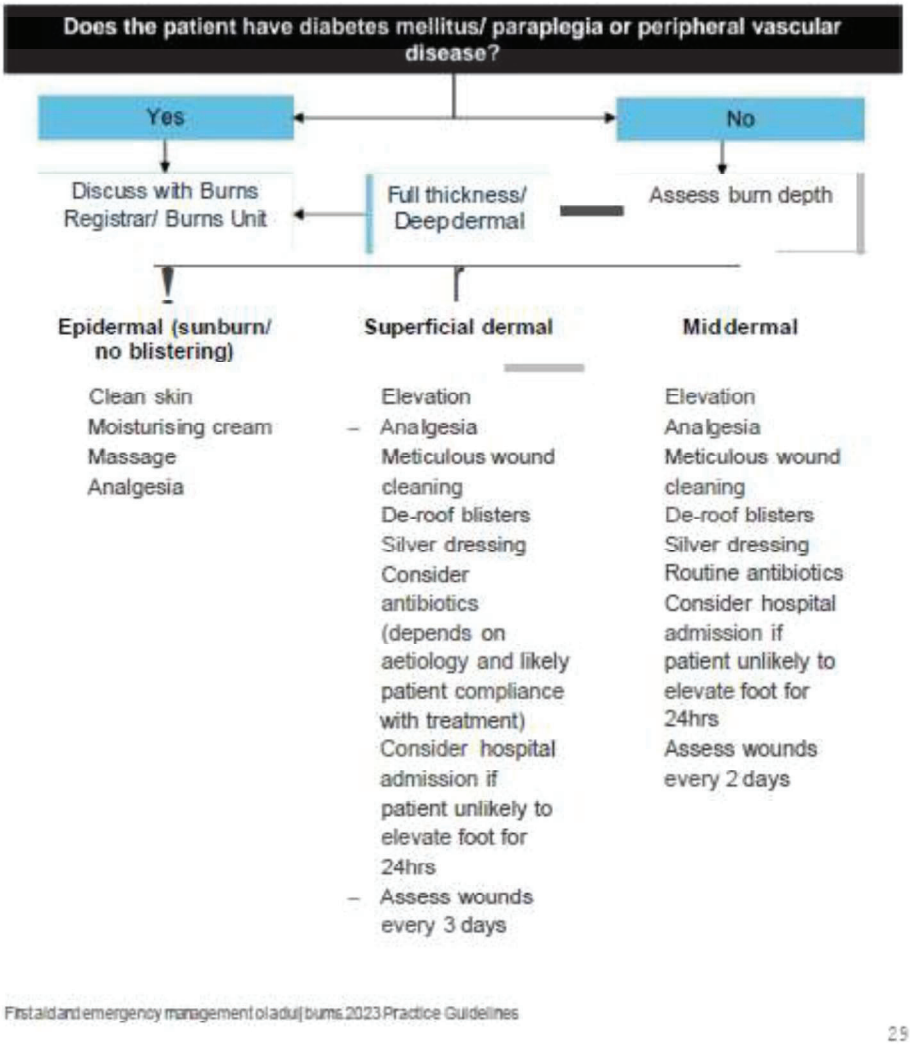
- Performed **6-hourly** with eye care (increase frequency if dry). Gentle cleaning and removal of existing paraffin and any newly declaring non-viable tissue. Forceps are to be avoided on the face.
  Application of a **thin** layer of white soft paraffin.
- **Performed 12-hourly** (with mouth and ear care) – male patients will undergo shaving of facial hair.
- **Performed daily** – hair washing.
- **Viral Swabbing** – Patients with facial burns (particularly those with a history of cold sores), virology swabs must be performed for Herpes Simplex Virus on days two and five **post-burn**. A positive result requires Famciclovir administration (herpes face burn infection is very painful, delays healing and leads to poorer healing and scarring).
- Patients with a strong history of HSV-1 and a facial burn should be considered for antiviral prophylaxis.

Appendix 0 Management of burns to the foot

Each foot is colonised by 1,000,000,000,000 bacteria. Inadequate management of foot burnsfrequently results inserious infection.This can lead to a need for skin grafting (where spontaneous healing was expected) and even digital/other amputation.

Avoid any constrictive/abrasive footwear - loose footwear should be worn. Initial elevationfor at least 24 hours is of utmost importance in preventing burn depth progression.

Time off work should be considered especially for those whose jobs entail standing or a hot,dusty dirty environment. As burns to the feet meet ANZBA referral criteria, consider contacting the Burns Unit/Burns Registrar for advice and how to best manage foot wounds .



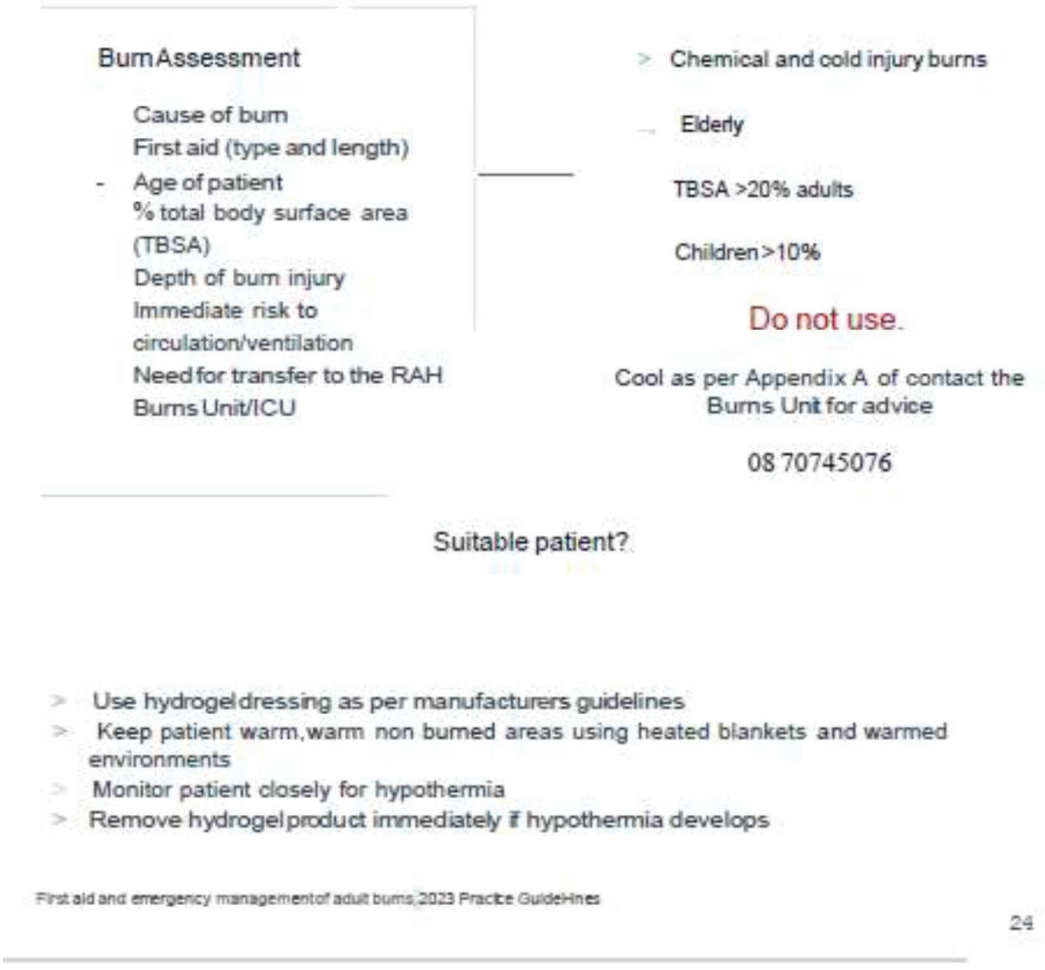
Appendix J - Protocol for the use of Hydrogel cooling products in burn injury first aid

Evidence indicates that the cooling function of Hydrogel dressing products is not as effective as cool running water.

If no clean water available,application of Hydrogel may be useful as an analgesic but should be replaced by water as soon as available if within 3 hours of injury.

Patients with extensive burn wounds (>20% TBSA in adults or >10% TBSA in children) are at increased risk of hypothermia

Hydrogels,or any wet dressing, can also be associated with the development of hypothermia if exposed to the air and left in place for prolonged periods, particularly in the elderly or children with larger burns and SHOULD BE AVOIDED ANZBA Consensus Statement· Firat aid and the use of Hydrogel&, Aug 2021


Burn Assessment
Cause of burn
First aid (type and length)
Age of patient
% total body surface area (TBSA)
Depth of burn injury
Immediate risk to circulation/ventilation
Need for transfer to the RAH Burns Unit/ICU
Chemical and cold injury burns
Elderly
TBSA >20% adults
Children >10%
Do not use.
Cool as per Appendix A of contact the Burns Unit for advice
08 70745076
Suitable patient?
Use hydrogel dressing as per manufacturers guidelines
Keep patient warm,warm non burned areas using heated blankets and warmed environments
Monitor patient closely for hypothermia
Remove hydrogel product immediately if hypothermia develops
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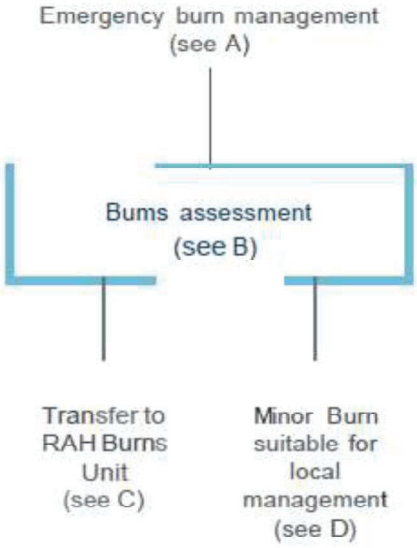
Appendix K - Primary burn wound care guidelines - adults



A. Emergency Management
Refer RAH first aid and emergency management guidelines

B. Bum AaMument
- cause of bum
- First aid (type & length)
- Depth
- %totalbody surface area (TBSA)
- Site of bum
- ventilation
- Need for transfer/ consultation* to RAH Burns Unit

C.Transfer to RAH Burns Unit
Anticipated time to arrivalat RAH
<1hour
- Face- wet soaks
- Other burn areas- cling film

1 4 hours
- Face- wet soaks
- Other burn areas- cling film

4-24 hours
- Face- soft paraffin
- Other burn areas- Jelonet ™, Bactegras ™or Inadine ™

>24 hours
- Face- soft paraffin
- Other burn areas- Jelonet™, Bactegras ™or Inadine ™
- Actlcoat™ following discussion w h receiving clinician (temporary skin staining arising from Act1coat ™ applicalion may make luture burn assessmenl / Biobrane application diff icult).

For any chemical injuries please contact the Burns Registrar through RAH switchboard (08) 70740000 for advice

D.Minor Burn for local management
Follow RAH dressing guidelines for minor burn management – Appendix F

Appendix P- Hydrofluoric acid burn treatment protocol (burns <2°/o TBSA and HF concentration <10%)

Copious irrigation for 30-120 mins to remove H'ion effect (burn).
Ends with patient's subjective cessation of 'burning' sensation.
If Diphoterine/Hexafluorine available,follow local guidelines

+

Apply calcium gluconate 2.5% gel to skin of entire burn area. Wash and reapply every 30 mins until resolution of pain, then every 4 hours
Clinician to wear double gloves

If primary survey passed,transport to RAH, if not consult nearest Trauma Centre

No deep tissue discomfort — Burns Unit Consultant and toxicobgy consult — Deep tissue discomfort (aching/ pain subcutaneously)

Wash and reapply gel every 30mins until cessation of pain,then every 4 hours consider removal of nails and application of gel to nail bed if affected.

Contact Burns consultant regarding further treatment
(08) 7074 5076

No deep tissue discomfort develops

Burns Unit admission overnight for observation

Appendix Q - Hydrofluoric acid burn treatment protocol (burns >2% TBSA and HF concentration >10%)

Patient at risk of systemic fluoride poisoning

Immediate Burns and Toxicology consultation

!

Local burn management as per protocol for <2% TBSA flow chart

!

VBG or ABG (check Ca^{2+} K+) MBA 20 and Mg - EGC

Patient stable and investigations normal

1

Six hourly ECG and venous gas
Twice daily MBA 20

Patient unstable or investigations abnormal

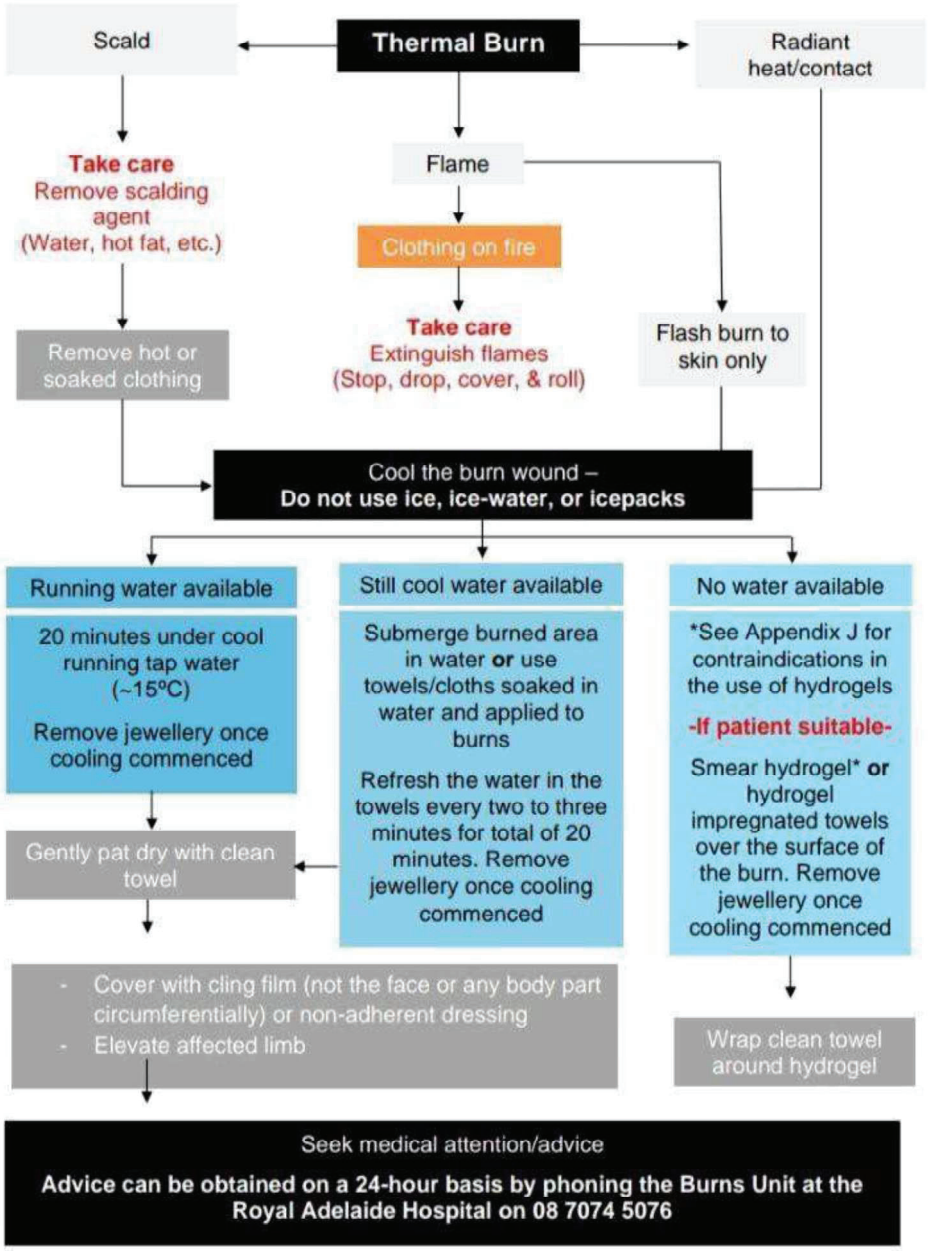
1

HDU/ICU

Aggressive replacement of Ca^{2+} and Mg^{2+}

Hourly VGB/ABG
Six hourly ECG, MBA 20 and Mg^{2+}

Appendix A – Community first aid protocol for thermal injury


Thermal Burn
Scald
Radiant heat/contact
Take care
Remove scalding agent
(Water, hot fat, etc.)
Flame
Clothing on fire
Take care
Extinguish flames
(Stop, drop, cover, & roll)
Flash burn to skin only
Remove hot or soaked clothing
Cool the burn wound –
Do not use ice, ice-water, or icepacks
Running water available
20 minutes under cool running tap water (~15ºC)
Remove jewellery once cooling commenced
Still cool water available
Submerge burned area in water or use towels/cloths soaked in water and applied to burns
Refresh the water in the towels every two to three minutes for total of 20 minutes. Remove jewellery once cooling commenced
No water available
*See Appendix J for contraindications in the use of hydrogels
-If patient suitable-
Smear hydrogel* or hydrogel impregnated towels over the surface of the burn. Remove jewellery once cooling commenced
Gently pat dry with clean towel
- Cover with cling film (not the face or any body part circumferentially) or non-adherent dressing
- Elevate affected limb
Wrap clean towel around hydrogel
Seek medical attention/advice
Advice can be obtained on a 24-hour basis by phoning the Burns Unit at the Royal Adelaide Hospital on 08 7074 5076

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SKIN TUMORS

Heng Monirath, Leng Sovannara

I. DEFINITION

Skin tumor are divided to 2 types:

- non-cancerous (benign) tumor of the skin is a growth or abnormal area on the skin that does not spread (metastasize) to other parts of the body.
- Skin cancer is the out-of-control growth of abnormal cells in the epidermis, the outermost skin layer, caused by unrepaired DNA damage that triggers mutations. These mutations lead the skin cells to multiply rapidly and form malignant tumors. The main types of skin cancer are Melanoma, Non - melanoma (Basal Cell Carcinoma (BCC), Squamous Cell Carcinoma (SCC)).

II. ETIOLOGY

- Benign skin tumor: Many benign skin growths are caused by years of exposure to the sun. Others run in families or appear to respond to hormonal changes. Pyogenic granulomas appear after damage to the skin from an injury.
- Skin cancer: The most commence are caused by the ultraviolet and radiation .

  The incidence of both non-melanoma and melanoma skin cancers has been increasing over the past decades. Currently, between 2 and 3 million non-melanoma skin cancers and 132,000 melanoma skin cancers occur globally each year. One in every three cancers diagnosed is a skin cancer and, according to Skin Cancer Foundation Statistics, one in every five Americans will develop skin cancer in their lifetime.

- Some individual risk factors for skin cancer
  - Fair skin
  - Blue, green or hazel eyes
  - Light-colored hair
  - Tendency to burn rather than suntan
  - History of severe sunburns
  - Many moles
  - Freckles
  - A family history of skin cancer

1. Bening skin tumor

Acrochordons

Acrochordons (skin tags) are extremely common, small, and typically

pedunculated benign neoplasms. They consist of hyperplastic soft dermis and epidermis, and are usually skin colored or brownish. They are generally 2 to 5 mm in size, although they may become larger. The most common locations are in skin folds (e.g., neck, axillae, groin), where skin irritation can be a causative factor. They occur in 25% to 46% of adults and increase with age and during pregnancy.[5] Studies have found that acrochordons are associated with the metabolic syndrome (obesity, dyslipidemia, hypertension, insulin resistance, and elevated C-reactive protein levels). This suggests they may be viewed as cutaneous clues for cardiovascular disease.

Acrochordons (skin tags) in a patient with metabolic syndrome.

Diagnosis is based on the appearance and location of lesions. They must be differentiated from neurofibromas, seborrheic keratoses, and pedunculated nevi. There have been rare case reports of skin tags that were found to be basal or squamous cell carcinomas. Treatment consists of cryosurgery, electrodesiccation, or simple scissor or shave excision. Electrodesiccation causes less hypopigmentation than cryotherapy and is the preferred treatment in nonwhite patients. An ear speculum placed over a small lesion may be helpful in directing the freeze pattern during cryosurgery.

Sebaceous Hyperplasia

Sebaceous hyperplasia is a benign disorder of the sebaceous glands that is common in middle-aged or older adults. Lesions present as asymptomatic, discrete, soft, pale yellow, shiny bumps on the forehead or cheeks, or near hair follicles. They typically appear as an umbilicated dome with multiple lobules resembling a cauliflower. There may be single or multiple lesions, ranging from 1 to 4 mm in diameter. They have no clinical significance except for cosmesis. Histologically, lesions consist of enlarged mature lobules of sebocytes around a central duct. It is important to rule out basal cell carcinoma, which is generally red or pink and increasing in size. Inspection of any surface vessels will show a haphazard arrangement in basal cell carcinoma, whereas the vessels in sebaceous hyperplasia occur only between lobules.

Sebaceous hyperplasia on the forehead with the typical umbilicated, lobulated appearance without haphazard blood vessels. No treatment is required for sebaceous hyperplasia, although patients may request removal of lesions for cosmetic reasons or because of concerns about malignancy. Therapeutic options include cryosurgery, phototherapy, shave excision, laser ablation, electrodesiccation with curettage, chemical cautery, or oral isotretinoin for widespread lesions.

Lipomas

Lipomas are slow-growing, benign mesenchymal tumors enclosed by a thin fibrous capsule. They closely resemble normal fat and are the most common

type of soft tissue tumor. They are usually subcutaneous but may occur in any organ because they are mesenchymal. They are generally asymptomatic but may become irritated with trauma or produce local obstructive symptoms in the airway or gastrointestinal tract. Their prevalence is 1%.[9]

Lipomas must be clinically differentiated from other tumors. The primary differential diagnosis in a subcutaneous mass is a sebaceous cyst or abscess. Lipomas are soft, flesh-colored nodules that are easily moveable under the overlying skin. Sebaceous cysts are generally identifiable by a central punctum, and abscesses can be identified by the presence of warmth, redness, and pain. Ultrasonography is increasingly used to aid in the diagnosis of lipomas. High-frequency ultrasonography (greater than 20 MHz) can provide high-resolution images of subcutaneous tumors and surrounding structures.[10] The differential diagnosis of lipomas also includes liposarcomas; risk factors for malignancy are size greater than 10 cm, older age, rapid lesion growth, location on the thigh, and invasion into deeper tissue, such as nerve or bone, leading to a firm or fixed feeling on examination. Lesions concerning for malignancy should be imaged with computed tomography or contrast magnetic resonance imaging.[11]

Patients commonly present with cosmetic concerns or symptoms related to compression of surrounding tissue. A single incision or punch excision (for smaller lesions) will generally allow manual expression of the lipoma without difficulty when standard excision is not required.[12]

Keratoacanthomas

Keratoacanthomas are rapidly growing, squamo proliferative benign tumors that resemble squamous cell carcinoma. They begin as round, firm reddish or skin-colored papules that develop into dome-shaped nodules with a keratin-filled crater. They may grow to 1 to 2 cm over weeks or months. There is a slower involution phase over several months, leaving a scar if not excised early in its course. Keratoacanthomas generally occur later in life on sun-exposed areas, primarily the face, arms, and legs. They are attributed to sun exposure, cigarette smoking, human papillomavirus infection, genetic factors, trauma, and chemical carcinogens.

Typical dome-shaped, keratin-filled plug of a keratoacanthoma on the dorsal arm.There is long-standing controversy over whether keratoacanthomas are benign, spontaneously self-limited tumors or a variant of cutaneous squamous cell carcinoma that have the potential for metastasis.[13] Keratoacanthomas share histopathologic characteristics that make them difficult to distinguish from squamous cell carcinoma. Shave biopsy may be inadequate to distinguish the conditions, whereas punch biopsy may be adequate because it obtains deeper tissue. Because no clinical or pathologic features can reliably differentiate keratoacanthoma from squamous cell carcinoma, early simple

excision of lesions is recommended, with margins of 3 to 5 mm. Mohs micrographic surgery may be considered if tissue sparing is desired.[14]

Medical treatment (systemic retinoids or intralesional injections of methotrexate, fluorouracil, or bleomycin) is reserved for nonsurgical candidates, patients with multiple lesions, and those with lesions on inoperable sites.

Pyogenic Granulomas

Pyogenic granulomas are rapidly growing nodules that bleed easily. Their name is a misnomer, however, as these lesions are neither pyogenic nor granulomas. They are an acquired benign tumor often found on mucous membranes. They tend to occur on the head or neck, or at sites of previous penetrating trauma. They are common in infancy and childhood, and approximately 2% of women develop a mucosal lesion in the late first to second trimester of pregnancy.

Pyogenic granulomas are yellow to purplish, pulpy vascular lesions often surrounded by a scaly collarette. They are usually removed because of their rapid growth and tendency to bleed. The differential diagnosis includes Spitz nevi, amelanotic melanoma, and squamous or basal cell carcinoma. Treatment options include shave excision with electrodesiccation of the base, and laser ablation.

Dermatofibromas

Dermatofibromas result from idiopathic benign proliferation of fibroblasts. Generally located on the lower extremities, they may develop at any cutaneous site and range in size from 3 to 10 mm. They are four times more common in women, and most develop between 20 and 50 years of age. They are usually asymptomatic, although pruritus and tenderness can be present. Dermatofibromas appear gradually over months and may persist for years. Although multiple dermatofibromas may be present, large numbers (15 or more) are rare. A multiple eruptive variant occurs in only 0.3% of patients, many of whom are immunocompromised (classically, those with human immunodeficiency virus infection or systemic lupus erythematosus).

Diagnosis is based on the appearance of firm, raised, papules or nodules, ranging from tan to reddish brown. They tend to be darker at the center and fade to normal skin color at the margin. Dermatofibromas exhibit dimpling or retraction of the lesion beneath the skin with lateral compression.

A typical dermatofibroma located on a lower extremity (A). The same dermatofibroma exhibits the dimple sign (B), a depression of the entire lesion (secondary to the dermal scarring seen as the major pathologic change) on lateral compression of the lesion with the thumb and index finger.

No treatment is required unless there is a change in size or color, or bleeding or irritation from trauma. A 2012 study found that 73% of patients who underwent laser ablation reported satisfaction with the results.

Epidermal Inclusion Cysts

Epidermal inclusion cysts are the most common type of cutaneous cyst. They are discrete nodules resulting from the implantation and proliferation of epidermal elements within the dermis. However, they display no sebaceous component and are not truly sebaceous cysts. They typically present on the head, neck, or trunk, and may remain stable or enlarge over time. Spontaneous inflammation and rupture can occur, with significant involvement of surrounding tissue. There is no way to predict which lesions will remain quiescent or become larger or inflamed. Infected cysts tend to be larger, more erythematous, and more painful than sterile inflamed cysts.

Multiple epidermal inclusion cysts are associated with Gardner syndrome, an autosomal dominant condition associated with colon cancer. Cysts that are unusual in number or location (e.g., fingers, toes) warrant screening for colon cancer. Malignancies (e.g., basal cell carcinoma, Bowen disease, squamous cell carcinoma, mycosis fungoides, melanoma in situ) can develop in cysts, but this is rare.

Diagnosis of epidermal inclusion cysts is based on appearance and palpation of a discrete, freely movable cyst or nodule. Careful inspection often reveals a central punctum. Treatment is unnecessary unless desired by the patient, and can be accomplished via simple excision with removal of the cyst and cyst wall. Inflamed or ruptured cysts often resolve spontaneously without therapy, although they tend to recur. Intralesional steroid injections can hasten resolution of inflamed cysts and should be followed by interval excision.23

Seborrheic Keratoses

Seborrheic keratoses are the most common benign epithelial tumor. They tend to be hereditary and occur after 30 years of age. They present as multiple, well-circumscribed, yellow to brown, raised lesions that feel slightly greasy, velvety, or warty and are described as having a “stuck-on” appearance

A well-circumscribed, yellow to brown, warty “stuck-on” seborrheic keratosis. The Leser-Trélat sign is the abrupt eruption of multiple seborrheic keratosis lesions in a patient with an underlying malignancy, usually an adenocarcinoma of the stomach. This is a rare sign supported mainly by case reports, but should prompt consideration of a paraneoplastic disorder. Seborrheic keratoses may resolve with treatment of the malignancy, then reappear with its recurrence. Seborrheic keratoses generally do not require treatment unless they become irritated or the patient has cosmetic concerns. They may be treated with

electrodesiccation, laser ablation, curettage, cryosurgery, or shave excision if biopsy is required.

Cherry Angiomas

Cherry angiomas are extremely common lesions that tend to appear with increasing age. They usually occur as multiple asymptomatic lesions, most commonly on the trunk and arms. They are dome-shaped, small (0.1 to 0.5 cm in diameter), bright red to violaceous, soft, compressible papules with smooth surfaces that blanch with pressure and bleed profusely with traumatic rupture. They can be treated effectively with electrodesiccation or laser ablation.

Table 1. Comparison of Common Benign Skin Tumors

Condition	Characteristics	Differential diagnosis	Treatment	Comments	Precautions and referral criteria
Acrochordon	Skin-colored to brown papules on narrow stalk	Senescent intradermal nevus	Cryosurgery, electrodesiccation, scissor or shave excision	Do not send multiple specimens in same jar	Cryosurgery should be performed with caution in persons with darker skin; refer patients with eyelid involvement
Cherry angioma	Dome-shaped, small, bright red to violaceous, soft, compressible papules	Pyogenic granuloma	Electrodesiccation, laser ablation	Numerous lesions (hundreds) and early onset can occur in Fabry disease	Genetic evaluation for Fabry disease in patients with multiple lesions
Dermatofibroma	Firm, raised, tan to reddish-brown papules or nodules; dimpling with lateral compression	Cellular dermatofibroma, dermatofibrosarcoma protuberans	Cryosurgery, intralesional steroid injection, laser ablation, punch excision	Abrupt appearance of multiple lesions may occur in persons with human immunodeficiency virus infection or systemic lupus erythematosus	Refer patients with cellular variant and dermatofibrosarcoma protuberans (deep invasion and metastases)
Epidermal inclusion cyst	Firm, mobile, subcutaneous nodule with central punctum; painless (unless inflamed)	Lipoma, abscess (vs. inflamed cyst)	Excision, intralesional steroid injection with incision and excision for inflamed cysts	Presence of punctum helps differentiate cysts from lipomas; history helps differentiate between inflamed cyst and abscess (acute)	Inflamed cysts and those that have undergone previous incision and drainage can be more difficult to excise; refer patients with facial cysts
Keratoacanthoma	Rapidly growing, dome-shaped hyperkeratotic papule on sun-damaged skin	Squamous cell carcinoma, verruca, hypertrophic actinic keratosis	Excision, intralesional injection (methotrexate, fluorouracil, bleomycin), Mohs micrographic surgery	Cannot be histologically differentiated from squamous cell carcinoma	Refer patients with recurrence after complete excision
Lipoma	Soft, mobile subcutaneous nodules	Epidermal inclusion cyst, liposarcoma, deep hemangioma	Incision or punch excision and manual expression	Ultrasonography can help differentiate lipomas from other deep neoplasms	Use caution with facial lipomas and recurrent lesions after excision
Pyogenic granuloma	Rapidly growing, yellow to violaceous, friable nodule, often surrounded by scaly collarette	Amelanotic melanoma, Spitz nevus, basal cell carcinoma, squamous cell carcinoma	Laser ablation; shave excision with electrodesiccation of base	Send for histologic evaluation to rule out melanoma	Refer patients with recurrent lesions or facial lesions
Sebaceous hyperplasia	Dome-shaped papule with central umbilication and uniform yellow lobules on magnification	Basal cell carcinoma	Chemical caustery, cryosurgery, electrodesiccation, laser ablation, oral isotretinoin, photodynamic therapy, shave excision	Thin shave biopsy can rule out basal cell carcinoma	Basal cell carcinoma is generally red or pink and increases in size
Seborrheic keratosis	Well-circumscribed, yellow to brown, "stuck-on" papules and plaques	Atypical nevus, melanoma	Cryosurgery, curettage, electrodesiccation, laser ablation, shave excision	Consider malignancy workup for abrupt appearance of multiple lesions	Cryosurgery should be performed with caution in persons with darker skin

a/k/a, Removed or excised lesions should be sent for histologic evaluation, especially pyogenic granuloma, lesions with atypical presentations, and lesions that recur after reasonable treatment (e.g., cryosurgery).

Table summary of benign skin tumor

Clinical subtypes of basal cell carcinoma

Subtypes of BCC	Clinical characteristics
Nodular	Glossy pearly papules or nodules with smooth surface and curled edges, dendritic capillaries, occurring on the head and neck (46-48)
Superficial	Well-defined and erythematous thin patches or plaques with scales, clear in the center and thinning at the edges. Common in the trunk area (48)
Micronodular	Erythema or thin papules/plaques
Infiltrative	Poorly defined, sclerotic, flat or depressed plaques that are white, yellow or pale pink and may be covered with crusts, erosions, ulcers or papules
Morpheaform	Infiltrative plaques with faint borders and shiny surfaces, commonly on the head and neck
Infundibulocystic	Well-defined pearly papules on the head and neck are common in the elderly
Fibroepithelial	Sessile patches of skin color or erythema or pedunculated papules with a predilection for the trunk (49)
Basosquamous	Most found on the head and neck (50)

BCC, basal cell carcinoma.

2. Malignancy of skin tumor

The diagnosis of the malignancy of skin tumor is result of histology.

a. Basal Cell Carcinoma

Low- and high-risk features of basal cell carcinomas

Grading criteria	Features[1]	Low risk BCC	High risk BCC
Clinical	Forms	Primary	Recurrent, metastatic
	Immune status	Immunocompetent	Immunosuppressed
	Anatomic location	Area L and M	Area H
	Radiotherapy	No	Yes
	Tumor boundaries	Well-defined	Poorly defined
	Tumor dimensions	Surface area[2]: area L, <20 mm; area M, <10 mm	Surface area[2]: area L, >20 mm; area M, >10 mm
		Size/diameter: <5 cm	Size/diameter: >5 cm
	Involvement of specified nerves	Absent	Present
Pathologic	Histologic type/growth pattern	Superficial, nodular, keratotic infundibulocystic, fibroepithelioma of Pinkus	Micronodular, infiltrative, sclerosing morpheaform, basosquamous, metatypical/sarcomatoid
	Perineural invasion	Absent	Present, diameter of involved nerve ≥0.1 mm, multifocality, involvement of named nerves

[1], features as defined by the National Comprehensive Cancer Network; [2], human skin is divided into three zones according to the risk of invasive keratinocyte carcinoma: area H is a high-risk zone (frontal hairline, central face, nose, eyelids, chin, ears, genitalia, hands, feet, and bald scalp); area M is a medium-risk zone (cheeks, forehead, scalp, neck, and jawline); and area L is a low-risk zone (trunk and extremities, excluding areas H and M). BCC, basal cell carcinoma.

Treatment options for basal cell carcinoma

Treatment	Indications	Advantages	Disadvantages	Histological assessment of excised tissue/margins
Surgical excision	Primary treatment option for small, well-defined tumors or large, low-risk tumors in low-risk areas	High cure rates	Potential for recurrence due to incomplete excision; contraindicated or impractical for some patients	Postoperative analysis
Mohs surgery	Treatment of choice for large, high-risk, recurrent, and facial tumors	Very high cure rate; preserves healthy tissue and minimizes scarring	Time-consuming procedure requiring specialized training	Intraoperative complete en face margin evaluation
Curettage and electrodessication	Small, low-risk, primary tumors	Inexpensive and fast procedure	Wounds heal slowly and cause scarring; high rate of recurrence with high-risk and recurrent tumors	Not assessed after treatment
Radiotherapy	Patients in whom surgery is contraindicated or impractical or based on patient considerations	Noninvasive, painless	Low cure rates vs (Mohs) surgery; generally reserved for older patients due to potential toxicities	Not applicable
Field therapies (topical 5-fluorouracil, imiquimod, photodynamic therapy)	Patients with low-risk, shallow, or superficial tumors contraindicated for surgery and radiotherapy	Typically, good cosmetic outcomes; inexpensive	Lower cure rates vs surgery and radiotherapy	Not applicable
HPI therapy	Adults with laBCC that is recurrent or who are not candidates for surgery and radiation or with mBCC	Efficacious in patients with advanced BCC	Not well tolerated in some patients due to low-grade AEs	Not applicable

Abbreviations: AE = adverse event, BCC = basal cell carcinoma, HPI = hedgehog pathway inhibitor, laBCC = locally advanced BCC, mBCC = metastatic BCC.

b. Squamous Cell Carcinoma

Cutaneous squamous-cell carcinoma can develop on any surface of the skin. It is more common in men than in women (3:1 ratio), and the risk increases dramatically with age. Specifically, the incidence among persons older than 75 years of age is 5 to 10 times that among those younger than 55 years of age. Patients typically present with scaly, erythematous, or bleeding

lesions, most often on sun- exposed areas, and the appearance of these lesions differs according to histologic subtype.

Table 1. Tumor Staging and Risk Factors for Cutaneous Squamous-Cell Carcinoma.

Tumor Stage	Staging System*				
	AJCC	BWH	Salamanca Refinement	Tübingen	NCCN
Low risk					
T1	Tumor diameter <2 cm	0 risk factors	Tumor diameter <2 cm	Tumor diameter ≤2 cm, tumor thickness ≤6 mm	Tumor diameter ≤2 cm on trunk and arms and legs; well-defined, primary tumor, well or moderately differentiated, depth ≤6 mm
T2	Tumor diameter ≥2 cm and <4 cm		Tumor diameter ≥2 cm and <4 cm		
T2a		1 risk factor			
High risk					
T2b		2 or 3 risk factors			**High risk**: tumor diameter >2 cm and ≤4 cm on trunk and arms and legs; location on head, neck, hands, or feet, pretibial area, or anogenital areas, regardless of diameter; poorly differentiated, recurrent, immunosuppression, site of prior radiation therapy or chronic inflammatory process, rapid growth, neurologic symptoms, perineural involvement. **Very high risk**: tumor diameter >4 cm at any location, poor differentiation, desmoplastic squamous-cell carcinoma, depth >6 mm or invasion below subcutaneous fat, perineural invasion of a nerve lying below dermis or measuring ≥0.1 mm, lymphatic or vascular invasion
T3	Tumor diameter ≥4 cm or minor bone erosion, perineural invasion, or deep invasion	≥4 high-risk factors or bone invasion			
T3a			Tumor thickness >6 mm (with no invasion beyond subcutaneous fat), with or without tumor diameter ≥4 cm		
T3b			Invasion beyond subcutaneous fat or perineural invasion		
T3c			Combination of both T3b risk factors or AJCC T3 definition with ≥3 risk factors		
T4a	Tumor with gross cortical bone or marrow invasion				
T4b	Tumor invading skull bone or involving skull base foramen				

* The American Joint Committee on Cancer (AJCC) *Cancer Staging Manual*, 8th edition, defines deep invasion as invasion beyond subcutaneous fat or at a depth of more than 6 mm. Perineural invasion is defined as invasion in nerves that are 0.1 mm or more in diameter, invasion that is deeper than the dermis, or clinical and radiologic involvement of affected nerves, without involvement or invasion of the base of the cranium. The Brigham and Women's Hospital (BWH) staging system defines high-risk tumors as having a diameter of 2 cm or greater, poorly differentiated histologic features, perineural invasion of 0.1 mm or more, or tumor invasion beyond subcutaneous fat (excluding bone invasion, which automatically upgrades the tumor to stage T3). NCCN denotes National Comprehensive Cancer Network.

Tumor Staging and Risk Factors for Cutaneous Squamous-Cell Carcinoma

Table 2. Guideline-Based Management and Risk-Adjusted Follow-up Recommendations for Primary Cutaneous Squamous-Cell Carcinoma.*

Risk Level	Surgery	Workup	Radiation Therapy	Systemic Therapy	Follow-Up
Low	Curettage and electrodesiccation (excluding terminal hair-bearing areas), wide local excision (margins, 4–6 mm), Mohs micrographic surgery	Clinical lymph-node evaluation	For patients who are not surgical candidates or who have positive margins	For patients who are not surgical candidates	Skin self-examination monthly; full-body skin examination every 3–12 mo for 2 yr, then every 6–12 mo for 3 yr, then annually
High	Mohs micrographic surgery or PDEMA preferred, wide local excision (margins, 5–10 mm)	Consideration of nodal staging (imaging) for BWH T2b or T3 tumors or AJCC T3 or higher-grade tumors	For patients who are not surgical candidates or who have positive margins, recurrent disease, or extensive or named-nerve perineural invasion	Multidisciplinary consideration of systemic therapy if there is residual disease after resection or if surgery is not feasible	Skin self-examination monthly; full-body skin examination plus clinical nodal examination every 3–6 mo for 2 yr, then every 6–12 mo for 3 yr, then annually
Very high	Mohs micrographic surgery or PDEMA preferred, wide local excision (margins, 5–10 mm)	Consideration of nodal staging (imaging or sentinel lymph-node biopsy)	For patients who are not surgical candidates or who have positive margins	Multidisciplinary consideration of systemic therapy alone in complicated cases of locally advanced squamous-cell carcinoma in which curative surgery or radiation therapy is not feasible	Skin self-examination monthly; full-body skin examination plus clinical nodal examination every 3–6 mo for 2 yr, then every 6 mo for 3 yr, then every 6–12 months

* PDEMA denotes peripheral and deep exhaustive margin assessment.

Guideline-Based Management and Risk-Adjusted Follow-up Recommendations for Primary Cutaneous Squamous-Cell Carcinoma.

c. Melanoma

Staging a melanoma provides a description of its size and whether it has spread to other parts of the body. The stage of your melanoma helps to guide your treatment. There are three ways to stage a melanoma, and all patients will undergo at least two of these:

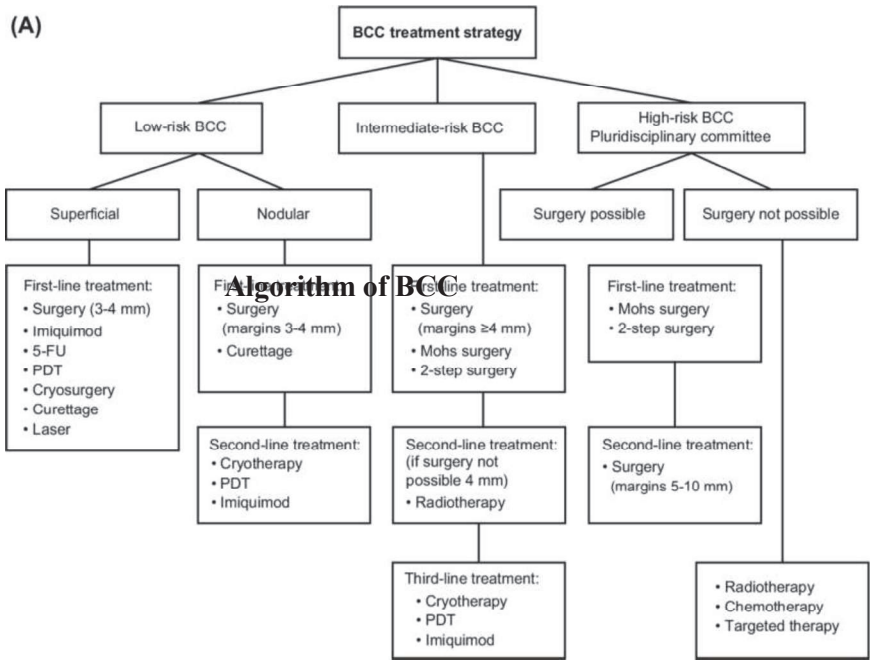
- **Microstaging –** A histopathologist looks closely at tissue under a microscope using special dyes and techniques to look for specific features that will help inform how far the disease has spread.
- **Clinical staging – The** lymph node groups that relate to the location of the melanoma are carefully examined to for any evidence of spread, usually seen by enlarged lymph nodes.
- **Staging after investigation –** This involves the use of imaging scans to see inside the body, including CT, MRI, and PET scans.

Stage	Definition	Main treatment options
In situ **Stage 0**	At this stage the tumour is confined to the cells in the top layer (epidermis) of the skin. The melanoma has not invaded deeper layers (dermis).	Surgical removal (wide local excision) is the main treatment.
Stage I	A Stage I melanoma can be up to 2 mm in thickness without ulceration; or up to 1 mm in thickness with ulceration.	Surgical removal is the main treatment. Sentinel lymph node biopsy (removal of nearby lymph nodes) may be considered to look for spread of melanoma to lymph nodes. It may be recommended if your melanoma is less than 1 mm in Breslow thickness or in some patients whose melanoma shows other adverse prognostic factors.
Stage II	Stage II melanoma is defined by thickness and ulceration. Tumours thicker than 2 mm with or without ulceration, and tumours between 1–2 mm with ulceration.	Surgical removal is the main treatment and sentinel lymph node biopsy may be considered to look for spread of melanoma to lymph nodes.
Stage III	A Stage III melanoma can be any thickness and has spread to nearby lymph nodes or tissues.	Surgical removal is the main treatment. Lymph node dissection (removal of all lymph nodes within the region concerned), drug and radiation therapies may be considered.
Stage IV	Stage IV melanoma can be any thickness, with spread (metastases) to distant lymph nodes or to distant sites (e.g. lung, liver, brain, bone).	Surgery or systemic (drug) therapies including immunotherapy and targeted therapy may be recommended. Radiation therapy may also be used.

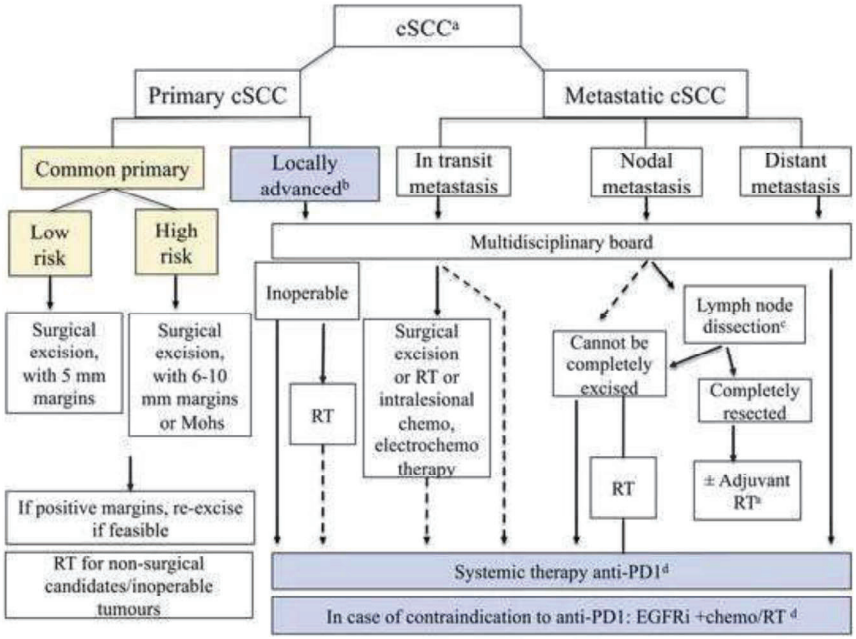
Management of melanoma

Stages	Definition	Main Treatment Options
In situ Stage 0	At this stage the tumour is confined to the cells in the top layer (epidermis) of the skin. The melanoma has not invaded deeper layers (dermis).	Surgical removal (wide local excision) is the main treatment.
Stage I	A Stage I melanoma can be up to 2 mm in thickness without ulceration; or up to 1 mm in thickness with ulceration.	Surgical removal is the main treatment. Sentinel lymph node biopsy (removal of nearby lymph nodes) may be considered to look for spread of melanoma to lymph nodes. It may be recommended if your melanoma is less than 1 mm in Breslow thickness or in some patients whose melanoma shows other adverse prognostic factors.
Stage II	Stage II melanoma is defined by thickness and ulceration. Tumours thicker than 2 mm with or without ulceration, and tumours between 1-2 mm with ulceration.	Surgical removal is the main treatment and sentinel lymph node biopsy may be considered to look for spread of melanoma to lymph nodes.
Stage III	A Stage III melanoma can be any thickness and has spread to nearby lymph nodes or tissues.	Surgical removal is the main treatment. Lymph node dissection (removal of all lymph nodes within the region concerned), drug and radiation therapies may be considered.
Stage IV	Stage IV melanoma can be any thickness, with spread (metastases) to distant lymph nodes or to distant sites (e.g. lung, liver, brain, bone).	Surgery or systemic (drug) therapies including immunotherapy and targeted therapy may be recommended. Radiation therapy may also be used.

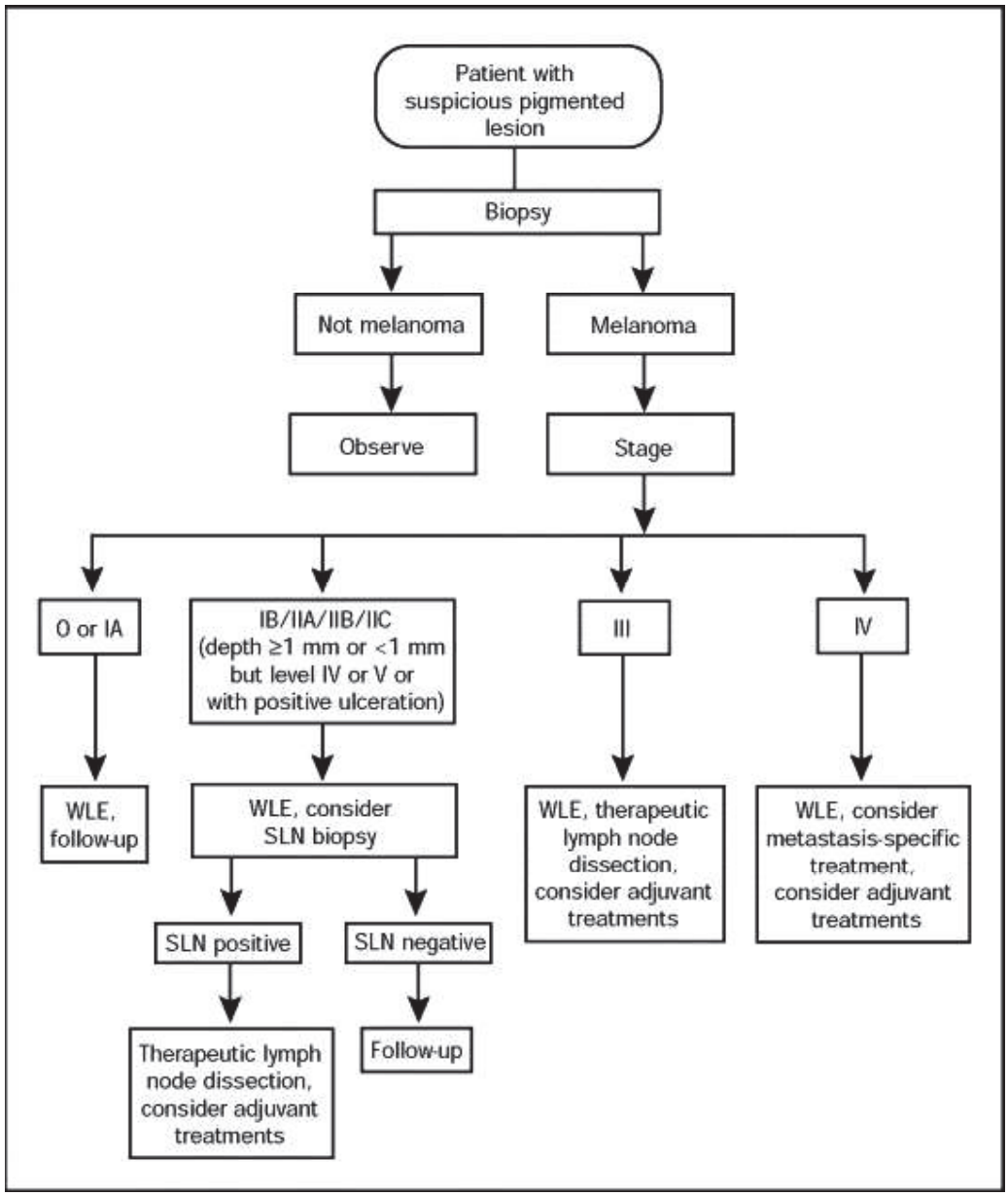
III. ALGORITHM



Algorithm of BCC



Algorithm of SCC treatment



Algorithm of melanoma treatment

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SOFT TISSUE INFECTION

KOEUT Kundara, MOK Theavy, Ky Chanmonyraksmey, Tep Borin, RY Sina, HENG Monyroth

I. DEFINITION

Soft Tissue Infections (STIs) are variety of pathological that involve underlying subcutaneous tissue, fascia, or muscle, ranging from simple superficial infections to severe necrotizing infections. STIs may affect any part of the body and are a frequent clinical problem in surgical departments.

Soft Tissue Infections (STIs) are classified necrotizing or non-necrotizing character of the infection, the anatomical extension, anatomic location, causative pathogen(s), rate of progression, depth of infection, and severity of clinical presentation. the characteristics of the infection (purulent or not purulent).

Simple abscess

2.1 Cutaneous abscesses are collections of pus within the dermis and deeper tissues. (induration and erythema only to a defined area of the abscess and should not have extension into deeper tissues or multiloculated extension).

2.2 Epidermoid cysts, often named "sebaceous cysts," are due to infection of pilosebaceous gland and are present anywhere in the body lined by squamous epithelium.

2.3 Furuncles are superficial infections with suppuration of the hair follicle, usually caused by *S. aureus*. They extend through the dermis into the subcutaneous tissue, where form a small abscess. Furuncles can occur anywhere on hairy skin. A group of infected hair follicles with pus is named Carbuncle.

2.4 Carbuncles are larger and deeper than furuncles. Furuncles often rupture and drain spontaneously or following treatment with moist heat.
A recurrent abscess at a site of previous infection may be caused by local causes such as a pilonidal cyst, hidradenitis suppurativa, or foreign material. Therefore, it always requires research of a local cause.

2.5 Treatment
Incision and drainage

Antibiotic therapy only in selected patients for 5 days. You may extend therapy up to 7–10 days if lack of symptom resolution at 5 days.
Incision, evacuation of pus and debris, and probing of the cavity to break up loculations provide effective treatment of cutaneous abscesses. **The pus and debris must be sent to lab for culture and antibiogram**. The resultant wound should be left open and lightly packed with roll gage soaked with antiseptic solution.
Antibiotic therapy should be prescribed for abscesses greater than 5 cm, in an area difficult to drain (e.g., face, hand, and genitalia), if there is lack of response to incision and drainage alone, if there are multiple localizations and in patients with immunosuppression.
Empiric antibiotic regimens. Normal renal function

Target Pathogens: *S.aureus* and streptococci.

- One of the following oral antibiotics
  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Trimethoprim and Sulfamethoxazole 160/800 mg every 12 h
  - Minocycline 100 mg every 12 h
  - Doxycycline 100 mg every 12 h
  - Amoxicillin-clavulanate 1 g every 8 h
  - Cephalexin 500 mg every 6 h
- In patients at risk for CA-MRSA including immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days or who do not respond to first-line therapy add one of the following oral antibiotics

  or
  - In patients with beta-lactam allergy
  - Clindamycin 300 mg every 8 h

In cases of recurrent skin abscess, it is necessary to look for the presence of foreign materials and identify and correct local factors that may cause recurring infection. For recurrent skin abscess bacterial cultureand antibiograme testing must be performed to verify the causative bacteria and antibiotics susceptibility to define a targeted therapy.

If an abscess is treated with prolonged antibiotics without drainage, it can lead to formation of sterile pus surrounded by thick fibrous tissue. It makes a hard lump that sometimes mimics malignancy. The treatment is surgical drainage with excision of fibrous wall.

1. Erysipelas

Erysipelas is a skin infection involving the dermis layer of the skin, but it may also extend to the superficial cutaneous lymphatics, a fiery red, tender, painful plaque with well-demarcated edges and is commonly caused by *Streptococcus spp.*, usually *S. pyogenes*. *S. aureus* rarely causes erysipelas.

Erysipelas is distinguished clinically from cellulitis by the following two features [1]:

- In erysipelas, the lesions are raised above the level of the surrounding skin, and
- Erysipelas is characterized by a clear line of demarcation between involved and uninvolved tissue.

Streptococci are the primary cause. The role of *S. aureus*, and specifically MRSA, remains controversial.

- Target Pathogens: *S. aureus* and streptococci, CA-MRSA is unusual.

Treatment

- Antibiotic therapy for 5 days. You may extend therapy up to 10 days if lack of symptom resolution at 5 days
- Use intravenous antibiotics if signs of systemic inflammation
- One of the following oral antibiotics:
  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Trimethoprim and Sulfamethoxazole 160/800 mg every 12 h
  - Minocycline 100 mg every 12 h
  - Doxycycline 100 mg every 12 h
  - Amoxicillin-clavulanate 1 g every 8 h
  - Cephalexin 500 mg every 6 h
  - In patients at risk for CA-MRSA including immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days or who do not respond to first-line therapy add one of the following oral antibiotics

    or
  - In patients with beta-lactam allergy
  - Clindamycin 300 mg every 8 h

or

Inpatient therapy

One of following intravenous antibiotics:

- Chloramphenicol: 12.5 mg per/kg every six hours.
- Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 12 h
- Linezolid 600 mg every 12 h
- Cefazolin 2 g every 8 h
- Amoxicillin-clavulanate 1.2/2.2 gr every 8 h

or

- In patients at risk for CA-MRSA including critically ill and immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days or who do not respond to first- line therapy add one of following intravenous antibiotics

Because of their very low yield, blood cultures are not helpful in managing erysipelas, unless it is particularly severe. Culture by aspiration or punch biopsy is not recommended as principle for identifying the causative bacteria in typical erysipelas patients. However, in several cases including immunosuppressed patients or those with

neutropenia, streptococci or *S. aureus* are not the causative bacteria. For these cases, lesion aspiration, or punch biopsy may be helpful to identify the causative bacteria and define a targeted therapy.

2. Cellulitis

Cellulitis is an acute bacterial infection primarily of the dermal lymphatics and the subcutaneous tissue that most commonly affects the lower extremities, although it can affect other areas. It causes local signs of inflammation, such as warmth, erythema, pain, lymphangitis, and frequently systemic upset impact with fever and raised white blood cell count. Outpatient therapy should be recommended for patients with adherence to therapy, who do not have general signs of inflammation or hemodynamic instability.

Patients with a previous attack of cellulitis, especially involving the legs, can present recurrences. The infection usually occurs in the same area as the previous episode. oedema, especially lymphedema, venous insufficiency, prior trauma (including surgery) to the area, and tinea pedis can increase the frequency of recurrences. Addressing these factors may decrease the frequency of recurrences.

The pathogens involved are streptococci and *S. aureus*. Cellulitis associated with abscesses is usually caused by *S. aureus*. In contrast, typical (non-purulent) cellulitis is most commonly caused by both streptococcal species and *S. aureus*. MRSA is an unusual cause of typical cellulitis.

In neutropenic and immunocompromised patients, Gram-negative bacteria should be considered.

Treatment

- Antibiotic therapy for 5 days or extend therapy up to 7-10 days if lack of symptom resolution at 5 days.
- Incision and drainage in purulent cellulitis and send pus to lab for culture and anti-biograme

2.1 Typical (non-purulent) cellulitis

Empiric antibiotic regimens. Normal renal function

- Target Pathogens: *S. aureus* and streptococci, CA-MRSA is unusual.

Outpatient therapy or step-down

- One of the following oral antibiotics
  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Trimethoprim and Sulfamethoxazole 160/800 mg every 12 h
  - Minocycline 100 mg every 12 h
  - Doxycycline 100 mg every 12 h
  - Amoxicillin-clavulanate 1 g every 8 h
  - Cephalexin 500 mg every 6 h
  - In patients at risk for CA-MRSA including immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days

or who do not respond to first-line therapy add one of the following oral antibiotics

or

- In patients with beta-lactam allergy
- Clindamycin 300 mg every 8 h

or

Inpatient therapy

- One of following intravenous antibiotics
  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 12 h
  - Linezolid 600 mg every 12 h
  - Cefazolin 2 g every 8 h
  - Amoxicillin-clavulanate 1.2/2.2 gr every 8 h

or

- In patients at risk for CA-MRSA including critically ill and immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days, with cellulitis associated with penetrating trauma especially from illicit drug use or who do not respond to first-line therapy one of the following intravenous antibiotics

- In patients at risk for Gram-negative infections or severe forms who do not respond to first-line therapy consider.

Piperacillin/tazobactam 4,5 g every 6 h.

2.2 Purulent cellulitis

Incision and drainage are recommended as primary management for abscesses with associated cellulitis. In these cases, antibiotics is generally suggested.

Empiric antibiotic regimens. Normal renal function

- Target Pathogen: *S. aureus* including CA-MRSA.

Outpatient therapy or step-down

- One of the following oral antibiotics

  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Trimethoprim and Sulfamethoxazole 160/800 mg every 12 h
  - Minocycline 100 mg every 12 h
  - Doxycycline 100 mg every 12 h
  - Amoxicillin-clavulanate 1 g every 8 h
  - Cephalexin 500 mg every 6 h
  - In patients at risk for CA-MRSA including immunocompromised status, personal or household contact with MRSA infection or colonization in the past 12 months, with prior antibiotic use for 5 days during the last 90 days or who do not respond to first-line therapy add one of the following oral antibiotics
    - or
  - In patients with beta-lactam allergy
  - Clindamycin 300 mg every 8 h

or

Inpatient therapy

- One of following intravenous antibiotics
  - Chloramphenicol: 12.5 mg per/kg every six hours.
  - Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 12 h
  - Linezolid 600 mg every 12 h
  - Cefazolin 2 g every 8 h
  - Amoxicillin-clavulanate 1.2/2.2 gr every 8 h

or

- In patients at risk for Gram-negative infections or severe forms who do not respond to first-line therapy consider
- Piperacillin/tazobactam 4,5 g every 6 h.

Culture is recommended as principle for identifying the causative bacteria in cellulitis patients. However, in immunosuppressed patients or those with neutropenia or in severe forms associated with systemic signs of inflammation or that do not respond to first-line therapy, culture may be helpful to identify the causative bacteria and define a targeted therapy.

Cellulitis in following situations can be life-threatening thus requiring early diagnosis and prompt intervention:

Orbital cellulitis: The infection usually spreads from paranasal sinuses and the patient presents with proptosis, chemosis, ophthalmoplegia and diminished vision due to pressure on optic nerve. Uncontrolled infection may have intra-cranial extension leading to cavernous sinus thrombosis and meningitis. Early intravenous empirical antibiotic therapy is required. Surgical drainage is required in progressive disease to prevent loss of vision.

Ludwigs angina: It is cellulitis of submandibular region occurring deep to deep cervical fascia leading to brawny induration in this region with edema of floor of the mouth. Untreated cases may have laryngeal edema and stridor. In case of no response to antibiotics, liberal fasciotomy of deep cervical fascia in the submandibular region is required.

3. Perianal and perirectal abscesses

Perianal and perirectal abscesses are typically well-circumscribed and respond to incision and drainage.Common sites of origin of complex abscesses may be perineal or perianal, perirectal. Antibiotic therapy should be used if systemic signs of infection are present, in immunocompromised patients, if source control is incomplete, or in cases of great abscess or with significant cellulitis. Empiric broad-spectrum antibiotic therapy with coverage of Gram-positive, Gram-negative, Klepsiella pheumoniae and anaerobic bacteria should be considered.

Diagnosis

In patients with perianal and perirectal abscesses, the diagnosis is often based on

clinical assessment and examination under anaesthesia. However, in some patients several imaging techniques may be useful.

1. EUS (anal endosonography)
2. CT

They should be evaluated according to the specific clinical scenario and the available technology and resources.

- Radiological studies are not usually needed to diagnose a complex abscess but can be useful in some special situations. The use of imaging techniques in perianal and perirectal abscesses could be helpful in all those cases with an atypical presentation (e.g., lower back pain, severe anal pain in the absence of a fissure, urinary retention), when the physical examination suggests a supra-elevator or inter-sphincteric abscess or when there is suspicion of perianal Crohn's disease or colonic source.

- MRI has high detection rates for anorectal abscesses [2], while there is debate around the sensitivity and specificity of (EUS). Some studies suggest that EUS is more accurate than MRI in detecting abscesses and evaluating complex fistulas, especially when suspected of arising from underlying Crohn's disease [3].

Treatment

- Incision and drainage + antibiotic therapy for 5 days in selected patients. You may extend therapy up to 7–10 days if lack of symptom resolution at 5 days.
- In perianal and perirectal abscesses identification of eventual fistula tract, and either proceed with primary fistulotomy to prevent recurrence (only in cases of low fistula not involving the sphincter muscle) or place a draining seton for future consideration. Fistulotomy can risk continence if too extensive and placement of seton should only be performed if the tract and openings are very clear, as there is risk of creating a false internal orifice and complicating the condition.

Empiric antibiotic regimens. Normal renal function

- Target Pathogen: Gram-positive, Gram-negative and Klepsiella pheumoniae

Outpatient therapy or step-down

- One of the following antibiotics
- Cefepime1 g every 8 h or
  - In patients with beta-lactam allergy
  - Ciprofloxacin 500 mg every 8 h + Metronidazole 500 mg every 8 h
  - In patients at risk for CA-MRSA or who do not respond to first-line therapy add one of following oral antibiotics
  - Minocycline 100 mg every 12 h
  - Trimethoprim and sulfamethoxazole 160/800–320/1600 mg every 12 h
  - Doxycycline 100 mg every 12 h or

Inpatient therapy

  - One of following intravenous antibiotics
  - Ceftriaxone 2 g every 24 h + Metronidazole 500 mg every 8 h
  - Cefotaxime 2 g every 8 h + Metronidazole 500 mg every 8 h
  - Piperacillin/tazobactam 4,5 g every 6 h

or

- In patients with beta-lactam allergy
- Ciprofloxacin 400 mg every 8 h + Metronidazole 500 mg every 8 h
- In patients at risk for CA-MRSA or who do not respond to first-line therapy add one of following intravenous antibiotics
- Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 12 h
- Linezolid 600 mg every 12 h

Culture is recommended as principle for identifying the causative bacteria in perianal or perirectal abscess. However, in immunosuppressed patients or those with neutropenia or in severe forms associated with systemic signs of inflammation or that do not respond to first-line therapy, it may be helpful to identify the causative bacteria and define a targeted therapy.

4. Necrotizing infections

NSTIs are life-threatening, invasive, soft-tissue infections with a necrotizing component involving any or all layers of the soft-tissue compartment, from the superficial dermis and subcutaneous tissue to the deeper fascia and muscle.

The vicious cycle of fulminant infection, toxin production, cytokine activation, micro thrombosis and ischemia, tissue dysfunction and death, and in turn, greater dissemination of infection is central to the rapidly progressive necrosis seen in NSTIs and differentiates it from that of the other SSTIs.

NSTIs have been described according to their anatomical locations (i.e., Fournier's gangrene) and the depth of infections: dermal and subcutaneous components (necrotizing cellulitis), fascial component (necrotizing fasciitis), and muscular components (necrotizing myositis).

Diagnosis

Conditions associated with NSTIs include diabetes mellitus, renal insufficiency, arterial occlusive disease, intravenous drug abuse, body mass index (BMI) > 30 kg/m^2, age < 65 years, liver disease, immunosuppression also in patients having tuberculosis and viral infections, recent surgery and traumatic wounds or incision of the skin, including minor lesions like insect bites and injections sites.

Diabetic patients exhibit impaired wound healing and increased susceptibility to infection, which may affect the course of SSTIs. It is thus reasonable to speculate that this chronic, debilitating disease contributes to a more serious nature of NSTIs. Diabetes mellitus is the most common co-morbidity associated with NSTIs. Up to 44.5% of patients with this condition are diabetic. Patients with diabetes generally present with polymicrobial and have poorer outcomes. Delay in diagnosis and delay in treatment of these infections increase the risk of mortality.

The initial differential diagnosis between cellulitis and NSTI that requires prompt operative intervention may be difficult. Most cases of NSTI are initially diagnosed as cellulitis. However, timely diagnosis is critical since time to operative debridement is an important determinant of outcome in NSTIs.

Patients with NSTI usually present with severe pain, which is out of proportion to the physical findings:

Local signs

- Edema
- Erythema
- Severe and crescendo pain out of proportion
- Skin bullae or necrosis (at a later stage)
- Swelling or tenderness
- Crepitus

The triad of swelling, erythema, and disproportionately severe pain should raise the suspicion of NSTI.

Systemic signs

- Fever
- Tachycardia
- Hypotension
- Shock

Laboratory tests are not highly sensitive or specific for NSTIs. A rapidly progressive soft-tissue infection should be treated as a necrotizing infection from the beginning. The clinical picture may worsen very quickly, sometimes during a few hours. But debridement necrotizing infection tissue culture or blood culture and antibiograme must be don early as possible.

To predict the presence of NSTI, the Laboratory Risk Indicator for Necrotizing infection (LRINEC) score was proposed (Table 1). LRINEC score assigns points for abnormalities in six independent variables: serum C-reactive protein level (> 150 mg/L), white blood cell (WBC) count (> 15,000/μL), hemoglobin level (< 13.5 g/dL), serum sodium level (< 135 mmol/L), serum creatinine level (> 1.6 mg/dL [142 mmol/l]), and serum glucose level (> 180 mg/dL [10 mmol/l]). With a score of 8 or higher, there is a 75% risk of an NSTI.

Table 1 Lrinec score *From: WSES/GAIS/WSIS/SIS-E/AAST global clinical pathways for patients with skin and soft tissue infections*

Variable (units)	Score points
C-reactive protein (CRP) (mg/L)	
< 150	0
≥ 150	4
White blood cell count (per mm^3)	

Variable (units)	Score points
< 15	0
15–25	1
> 25	2
Hemoglobin (g/dl)	
> 13.6	0
11–13.5	1
< 10.9	2
Serum sodium (mmol/L)	
≥ 135	0
< 135	2
Serum creatinine (mg/dl)	0
≤ 1.6	0
> 1.6	2
Serum glucose (mg/dl)	
≤ 180	0
> 180	1

Subsequent evaluation of the LRINEC score has demonstrated conflicting results. Several studies have assessed the utility of LRINEC for the early diagnosis of necrotizing infections.

Imaging

The diagnosis of NSTIs is primarily clinical. However, radiologic imaging may be able to provide useful information when the diagnosis is uncertain. A plain X-ray should not be used to rule out NSTI. However, it is important that if clinical suspicion of NSTI is high, radiologic imaging must neither delay nor deter surgery, because in this setting an early surgical debridement is essential to decrease mortality.

- Ultrasound
- CT

- MRI

CT has a higher sensitivity than plain radiography in identifying early NSTIs. Findings consistent with necrotizing infections are fat stranding, fluid and gas collections that dissect along fascial planes, and gas in the involved soft tissues. Additionally, fascial thickening and non-enhancing fascia on contrast CT suggests fascial necrosis.

MRI has been considered the imaging modality of choice for necrotizing fasciitis. However, MRI may be difficult to perform under emergency conditions and is not recommended as the first-choice imaging technique.

Treatment

- Surgical source control as soon as possible within 6 h after admission. Delay in early surgical increases mortality.
- Appropriate and effective debridement techniques. Skin-sparing debridement techniques focusing on tissue *directly involved in necrosis and send to laboratory culture and antibiograme.*
- Re-explorations should be repeated until the time when very little or no debridement is required.
- Empiric antibiotic therapy optimizing Pharmacokinetics (PK) and Pharmacodynamics (PD) targets.
- Deep samples collected at the interface between healthy and necrotized tissues during initial debridement and blood cultures allow the identification of causative pathogens in most cases.
- De-escalation of antibiotic therapy be based on clinical improvement, cultured pathogens, and results of rapid diagnostic tests where available.
- Hyperbaric oxygen therapy where it is available
- Intravenous immunoglobulin (IVIG) in patients with streptococcal NSTIs

Early source control, antibiotic therapy, and (organ) supportive measures are the cornerstone of treatment in patients with sepsis or septic shock caused by NSTIs.

Early surgical debridement with complete removal of necrotic tissue, including potential major amputation is essential to decrease mortality and other complications in patients with NSTIs.

It is the most important determinant of outcome in patients with NSTIs and should be performed as soon as possible, but at least within the first 6 h after admission.

Scheduled re-explorations should be done at least every 12–24 h after the initial operation or sooner if clinical local or systemic signs of worsening infection become evident, as well as with worsening laboratory parameters (WBC count, C Reactive Protein and Procalcitonin). Re-explorations should be repeated until the time when very little or no debridement is required.

After debridement and once the wound is stable, the subsequent use of negative pressure therapy allows reduction of the wound surface, extraction of wound exudate and cell residues, as well as induction of granulation.

Gas gangrene (clostridial myonecrosis), or type III NSTI, is an acute infection by clostridium or bacillus of healthy living tissue that occurs spontaneously or as a result of traumatic injury.

Occasionally in immunocompromised patients, NSTIs may also be caused by mycotic species. Empiric coverage against fungi should be started in high-risk patients. Since it is impossible to exclude with certainty a polymicrobial NSTI, an aggressive broad-spectrum empiric antimicrobial therapy should initially be selected to cover Gram-positive, Gram-negative, and anaerobic organisms until culture-specific results and sensitivities are available. An acceptable empiric antibiotic regimen should always include antibiotics, which cover MRSA with the additional benefit of inhibiting invasive hamolytic streptococci virulence proteins.

Selection of antibiotics that inhibit toxin production may be helpful, particularly in those patients who have evidence of toxic shock syndrome (TSS), potentially present in patients who have streptococcal and staphylococcal infections. Protein cytotoxins, such as superantigens, play an important role in the pathogenesis of various staphylococcal and streptococcal infections, and toxin production should be considered when selecting an antimicrobial agent for Gram-positive pathogens. Linezolid and clindamycin play an important role because they may significantly inhibit exotoxin production from Gram-positive pathogens [4]. Culture-specific results and sensitivities can direct both broadening of antibiotic regimen if it is too narrow and a de- escalation if it is too broad particularly in critically ill patients where de-escalation strategy is one of the cornerstones of antimicrobial stewardship programs [4].

In the absence of definitive clinical trials, antibiotic therapy should be administered until further debridement is no longer necessary. Patients with NSTIs are very complex and may lose fluids, proteins, and electrolytes through a large surgical wound. In addition, hypotension is caused by vasodilation induced by the systemic inflammatory response syndrome to infection and cytotoxins [5]. Fluid resuscitation and analgesia are the mainstays of support for patients with advanced sepsis, usually combined with vasoactive amines associated with mechanical ventilation and other organ function support, if needed. No ideal fluid has been proven: however, resuscitation therapy must be prompt and immediate as in any type of shock.

- As soon as possible after diagnosing sepsis (organ dysfunctions) associated with a NSTI, administer a 1-L bolus of a balanced crystalloid solution over 30 min. In hypotensive patients or those with an elevated serum lactate level additional fluid should be administered to achieve 30 ml/kg of initial volume resuscitation. This should be administered within 3 h.
- In patients who do not achieve a MAP $\geq$ 65 mmHg with initial volume resuscitation within one hour, start a norepinephrine infusion and titrate as needed. This can initially be administered through a peripheral IV while central venous access is being obtained.
- Simultaneously, administer within 1-h broad spectrum antimicrobial agent(s) to cover potential pathogens.
- If the norepinephrine infusion increases to $\geq$ 15 mg/min, add low dose vasopressin at infusion rate of 0.03 U/min. Do not increase this dose of vasopressin.
- Start low dose steroids (hydrocortisone 50 mg q 6 h) in patients requiring ongoing high doses of norepinephrine and vasopressin to achieve MAP $\geq$ 65 mmHg.
- Additional fluid resuscitation (beyond the initial 30 ml/kg) will likely be needed but should be based on the assessment that the patient will be fluid responsive.

- Patients with impaired cardiac function should inotropic agent started. Dobutamine is the preferred agent but will cause hypotension in hypovolemic patients.

New agents to treat NSTIs

Reltecimod (previously known as AB103 or p2TA), a peptide derived from the T-cell receptor CD28, modulates the host immune response by targeting the co-stimulatory pathway, which is essential for the induction of multiple pro-inflammatory cytokines. Consequently, reltecimod has demonstrated beneficial effects against different bacterial infections such as NSTIs.

A randomized, double-blind, placebo-controlled trial of single dose reltecimod (0.5 mg/kg) administered within 6 h of NSTI diagnosis was recently published [6]. Reltecimod was associated with improved resolution of organ dysfunction and hospital discharge status. Further studies are warranted to establish the real efficacy in clinical practice.

Wound management after source control

The rapidly spreading infection followed by aggressive surgical intervention and repeated debridement creates challenges for wound management.

Negative pressure wound therapy (NPWT) refers to wound dressing systems that continuously or intermittently apply sub-atmospheric pressure to the surface of a wound. NPWT has become a popular treatment modality for the management of many acute and chronic wounds [3]. In the setting of necrotizing infections once the necrosis is removed, NPWT can help wound healing physiologically. The negative pressure leads to an increased blood supply, increasing tissue perfusion, reducing edema, absorbing fluids and exudates, inhibiting infection, and finally drying the wound and thus the migration of inflammatory cells into the wound. Additionally, it promotes and accelerates the formation of granulation tissue by the removal of bacterial contamination and exudates. A modification of the original system added intermittent automated instillation of topical wound irrigation solutions to traditional NPWT.

Although skin grafting may fulfill this role, techniques higher on the reconstructive ladder, including local, regional and free flaps, are sometimes undertaken [7].

Empiric antibiotic regimens. Normal renal function

The initial empirical antibiotic regimen should comprise broad-spectrum drugs, including anti-MRSA and anti-Gram-negative coverage. Antitoxin active antibiotics such as clindamycin or linezolid should be included in the empirical antibiotic regimen to treat NSTIs.

In stable patients

- One of the following antibiotics
- Amoxicillin/clavulanate 1.2/2.2 g every 8 h
- Ceftriaxone 2 g every 24 h + Metronidazole 500 mg every 8 h
- Cefotaxime 2 g every 8 h + Metronidazole 500 mg every 8 h
- +

- Clindamycin 600–900 mg every 8 h

In unstable patients

- One of the following antibiotics
- Piperacillin/tazobactam 4.5 g every 6 h
- Meropenem 1 g every 8 h
- Imipenem/Cilastatin 500 mg every 6 h
- +
- One of the following antibiotics
- Linezolid 600 mg every 12 h
- Tedizolid 200 mg every 24 h or
- Another anti-MRSA-antibiotic as
- Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 8 h
- Daptomycin 6–8 mg/kg every 24 h *
- Telavancin 10 mg/kg every 24 h
- +
- Clindamycin 600–900 mg every 8 h

*Approved at the dosage of 4–6 mg/kg/24 h, it is currently used at higher dosages.

5. Fournier's gangrene

Fournier's gangrene (FG) is a severe type of NSTI involving the genital area and or perineum. The origin of the infection is identifiable in the majority of cases and is predominantly from anorectal, genito-urinary or local cutaneous sources. The aggressive nature of the infection requires early recognition and immediate surgical intervention.

Patients with FG usually present with severe pain, which is out of proportion to the physical findings

Diagnosis Local signs:

- edema.
- Erythema.
- Severe and crescendo pain out of proportion.
- Skin bullae or necrosis (at a later stage).
- Swelling or tenderness.
- Crepitus.

Systemic signs:

- Fever.
- Tachycardia.
- Hypotension.
- Shock.

Fournier's Gangrene severity index (FGSI) is a standard score for predicting outcome in patients with FG and is obtained from a combination of physiological parameters at admission, including temperature, heart rate, respiration rate, sodium, potassium, creatinine, leukocytes, haematocrit, and bicarbonate. An FGSI score above 9 has been demonstrated to be sensitive and specific as a mortality predictor in patients with Fournier's gangrene [8, 9, 10, 11] (Table 2).

Table 2 Fournier's Gangrene severity index

Physiological variables	+4	+3	+2	+1	0	+1	+2	+3	+4
Temperature (C)	> 41	39–40	–	38–39	36–38.4	34–35.9	32–33.9	30–31.9	< 29.9
Heart rate (bpm)	> 180	140–179	110–139	–	70–109	–	55–69	40–54	< 39
Respiratory rate	> 50	35–49	–	25–34	12–24	10–11	6–9	–	< 5
Serum K^+ (mmol/L)	> 7	6–6.9	–	5.5–5.9	3.5–5.4	3–3.4	2.5–2.9	–	< 2.5
Serum Na^+ (mmol/L)	> 180	160–179	155–159	150–154	130–149	–	120–129	110–119	< 110
Serum creatinine (mg/1000 ml) (× 2 for acute renal failure)	> 3.5	2–3.4	1.5–1.9	–	0.6–1.4	–	< 0.6	–	–
Hematocrit (%)	> 60	–	50–59	46–49	30–35	–	20–29	– < 20	
WBC (mm^3)	> 40	–	20–39.9	15–19	3–14.9	–	1.2.9	–	< 1
Serum bicarbonate venous (mmol/L)	> 52	41–51	–	32–40	22–31	–	18–21	15–17	< 15

Treatment

- Surgical source control as soon as possible. Re-explorations should be repeated until the time when very little or no debridement is required and send to lab for culture and antibiograms.
- Diverting colostomy or rectal diversion devices
- Antibiotic therapy
- (Organ) supportive measures

Surgical debridement must be early and aggressive to halt the progression of infection. Cultures of infected fluid and tissues should be obtained during the initial surgical debridement, and the results used to tailor specific antibiotic management. Radical surgical debridement of the entire affected area should be performed, continuing the debridement into the healthy-looking tissue [12, 13].

In the setting of FG, diverting colostomy has been demonstrated to improve outcomes but are poorly controlled studies that do not consider the morbidity and impact of the colostomy and its potential reversal.

A transverse loop colostomy is preferred because it yields solid and formed stools with little contamination of the surrounding skin. The abdomen above the umbilicus is ideal because FG often extends into the lower abdominal wall [14].

It may help in minimizing bacterial load in the perineal wound, thus controlling infection [15]. Diverting colostomy does not eliminate the necessity of multiple debridements nor reduces the number of these procedures [15]. Diverting colostomy should be avoided as much as possible, mainly when there are other methods to avoid wound contamination. Recently, rectal diversion devices have been marketed. They are silicone tubes designed to divert fecal matter in patients with diarrhoea, local burns, or skin ulcers. The devices protect the wounds from fecal contamination and reduce, in the same way a colostomy does, the risk of skin breakdown and repeated inoculation with colonic microbial flora. fecal diversion tubes can be used in combination with negative pressure wound therapy to effectively isolate the wound from fecal contamination [16]. Most FG infections are polymicrobial, and since it is impossible to exclude with certainty a polymicrobial necrotizing infection, an aggressive broad-spectrum empiric antimicrobial therapy should initially be selected to cover Gram-positive, Gram-negative, and anaerobic organisms until culture-specific results and sensitivities are available. An acceptable empiric antibiotic regimen should always include antibiotics, which cover MRSA with the additional benefit of inhibiting invasive GAS virulence proteins. For the treatment of MRSA, we refer to the previous paragraphs.

To treat Gram-negative bacteria, the use of piperacillin-tazobactam in the setting without the high local prevalence of ESBL-producing Enterobacteriaceae optimizing pharmacokinetic / pharmacodynamic parameters is appropriate. Carbapenems, administered in adequate dosage, including meropenem, imipenem-cilastatin, or doripenem, may be used in the settings with a high local prevalence of ESBL-producing Enterobacteriaceae. Culture-specific results and sensitivities can direct both broadenings of antibiotic regimen if it is too narrow and the de- escalation if it is too broad, particularly in critically ill patients where de-escalation strategy is one of the cornerstones of antimicrobial stewardship programs.

Empiric antibiotic regimens. Normal renal function

The initial empirical antibiotic regimen should comprise broad-spectrum drugs, including anti-MRSA and anti-Gram-negative coverage. Antitoxin active antibiotics such as clindamycin or linezolid should be included in the empirical antibiotic regimen to treat NSTIs.

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In unstable patients

- One of the following antibiotics
- Piperacillin/tazobactam 4.5 g every 6 h
- Meropenem 1 g every 8 h
- Imipenem/Cilastatin 500 mg every 6 h
- +
- One of the following antibiotics
- Linezolid 600 mg every 12 h
- Tedizolid 200 mg every 24 h

or

- Another anti-MRSA-antibiotic as
- Vancomycin 25–30 mg/kg loading dose then 15–20 mg/kg/dose every 8 h
- Teicoplanin LD 12 mg/kg 12-hourly for 3 doses, then 6 mg/kg every 12 h
- Daptomycin 6–8 mg/kg every 24 h *
- Telavancin 10 mg/kg every 24 h
- +
- Clindamycin 600–900 mg every 8 h

*Approved at the dosage of 4 mg/kg/24 h, it is currently used at higher dosages.

6. Gas gangrene

Gas gangrene also named clostridial myonecrosis and is another highly lethal NSTIs, caused by *Clostridium species*, with *Clostridium perfringens* being the most common [17].

Clostridial infections usually arise in traumatized tissues. However, it can also arise spontaneously. The infection involves deeper tissue such as a muscle which can lead to a rapidly spreading infection along tissue planes, and patients often present with sepsis.

C. perfringens causes 80–90%, of gas gangrene cases, but other species can cause infection, including *C. novyi, C. septicum, C. histolyticum, C. bifermentans, C. fallax,* and *C. sordellii* [17].

The fulminant clinical and histological features of an infection with clostridia are mediated by potent bacterial exotoxins, making clostridial myonecrosis the most rapidly spreading and lethal infection in humans.

Clostridium spp. can produce alpha and theta toxins that cause extensive tissue damage.

The primary toxin to mediate the effect of *C. perfringens* is alpha-toxin, a zinc metallophospholipase with phospholipase C and sphingomyelinase activity. Alpha-toxin is thought to be the major factor for tissue pathology leading to muscle necrosis and hemolysis [18]. The second major toxin is theta-toxin, a pore-forming toxin [19].

The infection can spread quickly, and within a matter of several hours, the patient may develop overwhelming shock, sepsis, and death. The infection can develop slowly over weeks or rapidly over hours depending on the oxygen tension of the tissue and the amount of organism inoculated.

Increasingly severe pain beginning within 24 h at the injury site is the first reliable clinical symptom. The skin may initially appear pale, but quickly changes to bronze, then purplish-red. The infected region becomes tense and tender, and bullae filled with reddish-blue fluid appear. Gas in the tissue, detected as crepitus or by imaging, is usually present by this late stage. Signs of systemic toxicity, including tachycardia, fever, and diaphoresis, develop rapidly, followed by shock and multiple organ failure.

Because the infection is rapidly progressive, it is important to treat patients aggressively, by early surgical debridement, antibiotics and intravenous fluid resuscitation.

Treatment / Management

Because the infection is rapidly progressive, it is important to treat patients aggressively with antibiotics, early surgical consultation with debridement, intravenous fluid resuscitation, ICU monitoring, and adjuvant hyperbaric oxygen therapy.

It is important to get early surgical consultation without delay as this is a true surgical emergency. Providers should not delay antibiotics to get cultures but should begin empiric treatment with antibiotics. Reasonable broad-spectrum coverage includes vancomycin and tazobactam or a carbapenem or ceftriaxone with metronidazole. If the provider suspects gas gangrene or a necrotizing soft tissue infection, then penicillin plus clindamycin should be added which will also treat group A streptococcal necrotizing fasciitis. Clindamycin should be strongly considered because it inhibits the synthesis of clostridial exotoxins and will lessen the systemic effects of these toxins. Because clindamycin is bacteriostatic and not bactericidal, it should be used in conjunction with a second anti-microbial such as penicillin.[20][21][22][23][24]

Fasciotomy may be necessary to relieve compartment pressures. As the infection progresses into deep tissue along and under the fascia tissue compartment pressures increase, which perpetuates further tissue ischemia and necrosis. Surgical debridement should focus on removing all the necrotic tissue, and foreign bodies such as soil, debris, and shrapnel. It is also important to irrigate the wounds with copious amounts of sterile normal saline.

Hyperbaric oxygen therapy should be added to standard therapy of antibiotics and surgical debridement to help improve survival.[24][25] It is important to have coordinated care of these critically ill patients with an intensivist, general surgeon, orthopedic surgeon, urologist (in the setting of Fournier's gangrene of the testicles and perineal structures), gynecologist (in the setting of uterine gas gangrene),
infectious disease specialist, hematologist/oncologist, gastroenterologist (in the setting of spontaneous gas gangrene), and hyperbaric oxygen therapy specialist. The flow of consultation starts with usually an emergency department provider and early recognition of the disease.[26][27][26]

Early IV antibiotics with early surgical debridement followed by hyperbaric oxygen therapy can salvage patients with an otherwise nearly always fatal disease. Intravenous antibiotics and early surgical debridement of the necrotic tissue reduce the fatality rate to about 30%. With the addition of hyperbaric oxygen therapy, this can
be reduced down to 5 to 10%. Hyperbaric oxygen therapy helps by halting exotoxin production by the bacteria, helps to improve the bactericidal effect of the antibiotic, treats the tissue ischemia, improves reperfusion injury of the tissue, and promotes the activation and migration of stem cells and polymorphonuclear cells. Additionally, hyperbaric

oxygen induces vasoconstriction reducing tissue edema, while augmenting oxygenation. The oxygen tension of the tissue increases by a factor of 1000 and this increased oxygen in the tissue helps to resolve hypoxia, improve cellular activity, inhibit bacterial growth, and affect cytokinesis that increases migration of neutrophils to the injured tissue. Hyperbaric oxygen also increases the production of growth factors such as vascular epidermal growth factor (VEGF) which induces neovascularization and tissue repair with capillary budding. This is recognized clinically as increased granulation tissue formation and is usually seen after several hyperbaric oxygen treatments.[26][29][30][29]

Hyperbaric oxygen therapy involves placing the patient in a pressurized chamber which can be mono-place (single patient) or multi-place (multiple patients treated at the same time). The mono-place chamber can only treat one patient at a time, and the attendant is outside of the chamber with specialized equipment and pumps to run IVs and even mechanical ventilation equipment through ports in the chamber door or wall. The disadvantage of this setup is that it limits the therapies available in the chamber and if the patient requires direct contact with the attendant, the chamber has to be depressurized, and the patient is taken out of the chamber. The multi-place chamber has the added benefit of being able to treat multiple patients at the same time, and the attendant is in the chamber with the patients allowing easier access to the patient for ventilator support, IV therapy, placement of a chest tube, or needle decompression of a pneumothorax. The treatment pressure for gas gangrene is 3 atmospheres absolute (ATA). The patient will have air brakes about every half hour to help reduce the risk of oxygen toxicity. These air brakes are usually 5 to 10 minutes in duration. The total duration of the treatment at pressure is usually about 90 minutes with 10 minutes for descent and 10 minutes for the ascent.[22][31][32][22]

When treating gas gangrene, the treatments start twice a day for the first 5 to 10 treatments, reducing to once-daily treatments when stabilized. Continuing hyperbaric oxygen therapy beyond the initial stabilization can speed healing of tissue and preparation for eventual tissue grafting that is often necessary to close the large defects left after surgical debridement of dead tissue. The risk of hyperbaric oxygen therapy includes oxygen toxicity which can cause seizures, hypoglycaemia especially in insulin- dependent diabetics, and barotrauma which can affect the ears, lungs, or any gas-filled structures, such as the stomach, and gas embolism. These complications are rare, except for ear barotrauma which occurs approximately 43% of the time (84% of these are minor injections of the tympanic membrane).

It is crucial to get early surgical consultation without delay in the case of gas gangrene, as this is an immediate emergency. Broad-spectrum antibiotics should be initiated without any delay to get cultures. Reasonable coverage should include vancomycin, tazobactam or a carbapenem, or a third-generation cephalosporin (ceftriaxone) with metronidazole. Moreover, in case of any suspicion for gas gangrene or necrotizing fascitis, penicillin plus clindamycin should be added to cover group A

streptococcal necrotizing fasciitis. Clindamycin is strongly recommended. Adjunctive measures in the treatment of gas gangrene include hyperbaric oxygen (HBO) therapy. The function of the existing toxin is not affected by hyperbaric oxygen therapy; thus, debridement is of paramount importance. Hemodynamically unstable patients may not be candidates for HBO therapy. Moreover, animal experimental studies in animals failed to document the therapeutic efficacy of HBO.[32][20]

Providers should consider the use of negative pressure wound dressing therapy once adequate surgical debridement has resolved ongoing tissue necrosis.

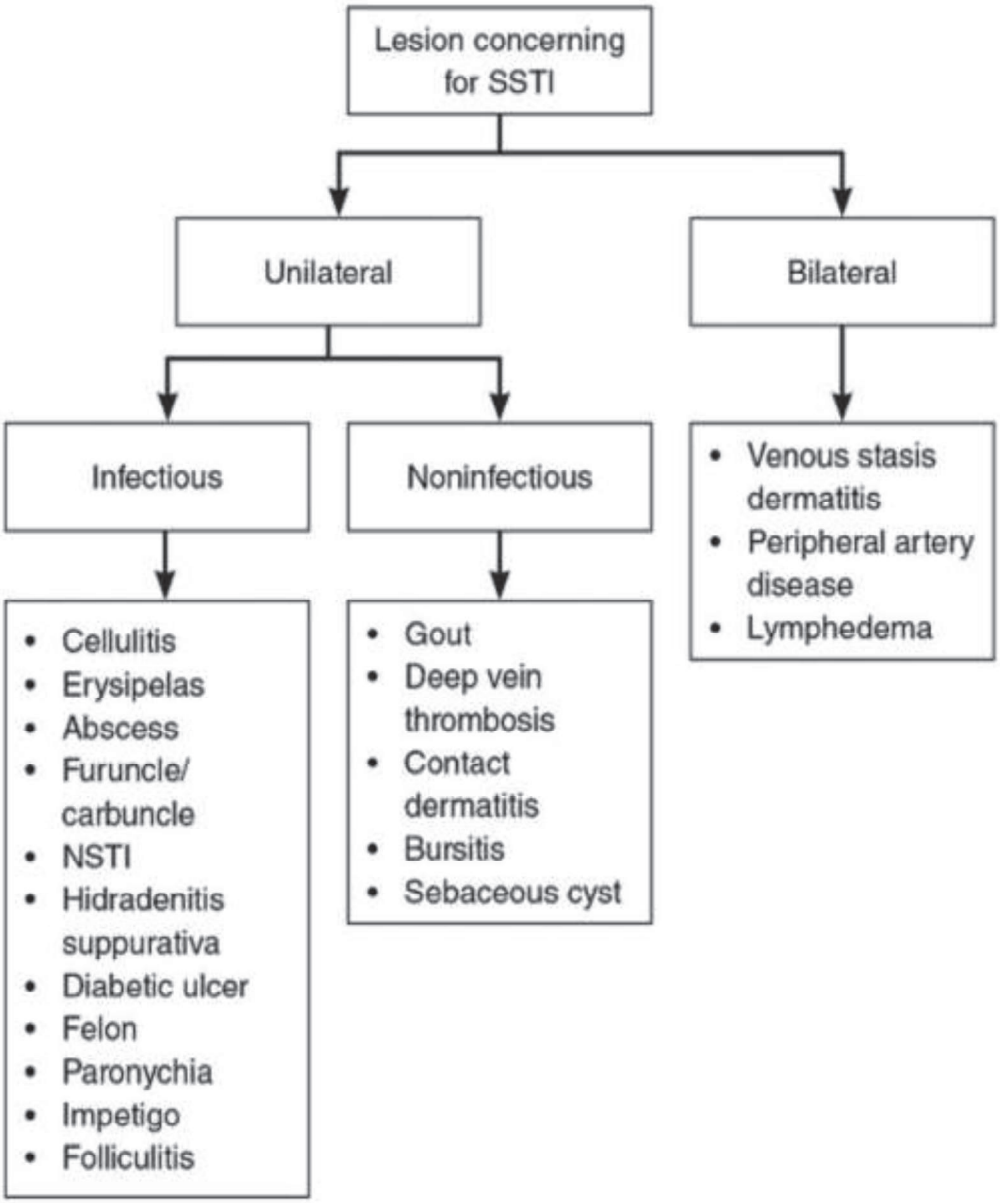
Table: clinical practice and CPA

Pathology	**Clinical signs**		**Bacteria**	**Indication**	**Anest**	**CPA**
Simple abscess	Skin abscess	Collection pus dermis and deep tissues, induration, Erythema	Sta. aureus, E.colie, Stre.pyogenes	Inci. drainage + open wound	LRA GA	CPA 2,3
	Epidermoid cysts infected	Or Sebaceous cysts Infection pilo-sebaceous gland: Lumps or bumps warmth tenderness and redness	Coci grame +, Stap, Anaerobie Fungus	Removal including capsule + open	LRA	CPA 2,3
	Furuncles	Superficial Infec. with suppuration of hair follicle: redish tender bumps + pus	Staphylococcus aureus	Inci drainage + open	LA	CPA 2,3
	Carbuncles	larger and deeper than furuncles or Clusters of Furuncles and fever	Staphylococcus aureus	Inci drainage + open	LA/GA LRA	CPA 2,3
Erysipelas	skin infection involving the dermis layer, but it may also extend to the superficial cutaneous lymphatics: fiery red, tender, painful plaque with well-demarcat- ed edges. distinguished clinically from cellulitis by the following two features : • the lesions are raised above the level of the surrounding skin, • Erysipelas is characterized by a clear line of demarcation between involved and uninvolved tissue.		Streptococcus spp., usually Staphylococcus pyogenes. Staphylococcus aureus	Medical treament		CPA 1,2,3
Cellulitis	infection primarily of the dermal lymphatics and the subcutaneous tissue. warmth, erythema, pain, lymphangitis, and frequently systemic upset impact with fever		Streptococcal species and *S. aureus*. Gram -	Incision drainag / Faciotomy + open wound	LRA GA	CPA 2,3

Perianal and perirectal abscesses	Anal pain, Fever, Chills, Constipation, or diarrhea Purulent discharge, Erythema and iduration in the skin around the perianal.	Gram-positive, Gram-negative, and anaerobic bacteria	Incision drainag + open wound	LRA GA	CPA 2,3
Necrotizing infection	Aggressive invasive, soft-tissue infections with a necrotizing component involving any or all layers of the soft-tissue compartment, from the superficial dermis and subcutaneous tissue to the deeper fascia and muscle. Local signs: - Edema, Erythema - Sever pains - Skin bullare or necrosis Swelling or tenderness - Crepitus Systemic signs: - Fever - Tachycardia - Hypotension - Shock	Staphylococcu Streptococcus A Polymicrobial - Gram (- and +), and anaerobic - Clostridium myonecrosis - Bacterioides coliforms, prot eus, klebsiella, peptostreptoc oc cus and pseudomona s	- Debridement surgical as soon as possible within 6 h after admission. Re-explorations should be done at least every 12–24 h after ini tial operation or sooner if clinical local/systemi c signs worsening	LRA GA	CPA 3
Fournier's gangrene	is a severe type of NSTI involving the genital area and or perineum. Local signs: - Edema, Erythema - Sever and crescendo pains - Skin bullare or necrosis - Swelling or tenderness - Crepitus Systemic signs: - Fever - Tachycardia - Hypotension - Shock	Staphylococcu Streptococcus A Polymicrobial - Gram (- and +), and anaerobic - Clostridium myonecrosis - E-coli and pseudomona s	- Debridement surgical as soon as possible within 6 h after admission. Re-explorations should be done at least every 12–24 h after ini tial operation or sooner if clinical local/systemi c signs worsening	LRA, SP/RA GA	CPA 2,3

			- Colostomy or rectal diversion devices		
Gas gangrene	- named clostridial myonecrosis and Clostridial infections usually arise in traumatized tissues. - spread quickly of several hours. - patient may develop overwhelming shock, sepsis, and death. - severe pain beginning within 24 h at the injury site. - skin may initially appear pale, but quickly changes to bronze, then purplish-red. - infected region becomes tense and tender, and bullae filled with reddish-blue fluid appear. - Signs of systemic toxicity, including tachycardia, fever, and diaphoresis, develop rapidly, followed by shock and multiple organ failure.	*Clostridium species Clostridium perfringens* causes 80–90%, but other species can cause infection, including *C. novyi, C. septicum, C. histolyticum, C. bifermentans, C. fallax,* and *C. sordellii*	- Debridement / Fasciotomy surgical as soon as possible. - Hyperbaric oxygen therapy	LRA SA/RA GA	CPA 3

DIFFERENTIAL DIAGNOSES OF SOFT-TISSUE INFECTION



Abbreviations: NSTI, necrotizing soft-tissue infection; SSTI, skin and soft-tissue infection.

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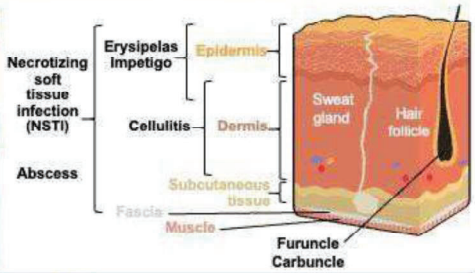
SKIN AND SOFT TISSUE INFECTIONS

SIGNS & SYMPTOMS

- Erythema, warmth, tenderness, pain, fever, purulence
- **Systemic signs:** Temp. >38°C, HR >90, RR >24 or WBC <12,000 or <300 cells/uL

HISTORY

- Onset of signs/symptoms? Progression?
- Association with trauma?
- Burn(s), frostbite, pressure ulcer, post-surgical?
- Environmental risks? Vaccination history?
- Severity of pain? Radiation?
- Loss of function? Joint involvement?



Non-Purulent SSTI			
Erysipelas	Associated with fever, well demarcated erythema	*Group A Streptococcus* (GAS)	
Impetigo	**Non-bullous:** painless, erythematous base w/ honey-crusted exudate on face/limbs	*S. aureus*, GAS	
	Bullous: clusters of bullae/solidary lesions of exudate ± desquamation	Toxin producing *S. aureus*	
Cellulitis	Edema, pain, poorly demarcated erythema. Orbital cellulitis is a medical emergency!	GAS, *S. aureus*	
Necrotizing Soft Tissue Infection (NSTI)	Generally, rapidly evolving, pain out of proportion, erythematous rash w/ fever, toxic appearance, & thrombocytopenia. Hemodynamically unstable.	**Monomicrobial** *S. aureus* most common	**Polymicrobial** GAS, MRSA, VRE, *Clostridium*
Purulent SSTI (Drainable Collection)			
Cutaneous / Deep Soft Tissue Abscess	Collection of pus	*S. aureus* (community-acquired MRSA), GAS	
Furuncles, Carbuncles ± Cellulitis	**Furuncles (boils)** are skin abscesses that involve a hair follicle. **Carbuncles** are clusters of furuncles.	*S. aureus* (including MRSA), GAS	

Mild: cellulitis, erysipelas, ± purulence
Moderate: above w/ systemic signs
Severe: failed oral antibiotics, systemic symptoms, immunocompromised, signs of deeper infection (bullae, skin sloughing, hypotension, organ dysfunction)

Cellulitis, Erysipelas, Necrotizing infection

MILD
Oral
Cephalexin
Cloxacillin

MODERATE
Intravenous
Cefazolin *OR*
Cloxacillin *OR*
Ceftriaxone *OR*

SEVERE
Emergent surgical inspection, debridement
R/o necrotizing process
Empiric Rx:
1) Vancomycin
2) ***PLUS*** Piperacillin-Tazobactam
3) ***PLUS*** Clindamycin*

C&S for definitive management

Furuncle, Carbuncle, Abscess

MILD
Incision and Debridement (I&D)

MODERATE
I&D *PLUS*
Cephalexin PO *OR*
Cefazolin IV

SEVERE
I&D *PLUS*
Vancomycin IV

C&S of purulent fluid for definitive management

NSTIs IN PEDIATRICS

1) No skin manifestations in up to 50% of cases.
2) A prolonged prodrome or perceived slow onset should NOT rule out an NSTI.
3) May present with multifocal sites. Always check for additional sites during the physical exam.

EAGLE EFFECT

The **Eagle Effect** is the paradoxical effect of reduced penicillin efficacy at higher antibiotic doses. **Clindamycin** also inhibits bacterial toxin production.

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